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PROGRESSIVE DEAFNESS, OTOSCLEROSIS AND CLOSELY RELATED SUBJECTS. AN ABSTRACT OF THE AVAILABLE LITERATURE PUBLISHED DURING THE YEAR 1940.

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International events have greatly limited the number of contributions of value upon progressive deafness and otosclerosis during the past year. Little has been presented from Continental Europe for obvious reasons, and there has been a definite limitation upon the splendid work usually presented from the British Empire.

In America much of the presentation has been analytical rather than constructive, and that which has concerned medical and surgical therapeusis, while definitely controversial, has, nevertheless, led to a better understanding of their present status.

More and more attention appears to have been paid to national efforts both in the prevention of deafness and in the conservation of hearing. These have apparently absorbed so much attention that real pathologic study has remained somewhat in the background. Much interest has centered upon the precision in audiometric standards and the accurate prescription of hearing aids. Much has been eliminated that has proved to be without great promise in specialized medication; for example, prostigmin, the estrogenic substances, thyroxin, etc. Far more uniformity in acceptance of fistulization standards has also occurred and the confusion of reference in the

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terms progressive deafness and otosclerosis has been clarified, though precisional directions and safeguards in the use of the new remedies have pertained.

Striking progress during the year would seem to have been made in the tests for loudness balance, both in reference to the masking of tinnitus and the discovery of nerve deafness; the clarification of the principles brought forth by Crowe and his associates on the management of lymphoidal tissue in the nasopharynx and the Eustachian tube; precisional understanding in the use of the newer biochemical remedies, and Covell's studies in avitaminosis.

In the articles abstracted, the author's opinions have been reported without individual comment. As these contributions have presented rather classic features, for convenience the following grouping has been followed:

Group 1. Clinical Studies and Research.

Group 2. Medical Therapeusis and Systemic Readjustment.

Group 3. Mechanical and Surgical Procedures.

Your Editors have found it difficult to more clearly comment on the progress of the year in this field and must leave the decision to your perusal of the abstracted material. As far as feasible, the articles have been arranged in chronologic order.

GROUP 1. CLINICAL STUDIES AND RESEARCH.

Fowler[1] presented "Head Noises. Significance, Measurement and Importance in Diagnosis and Treatment," following his previously reported observations on the loudness of tinnitus and its rôle in accentuating deafness. Last year the masking of tinnitus was reported, in which cases were demonstrated by audiograms showing tinnitus in different frequency bands, masked by pure tones at or slightly above the intensity of the tinnitus. Also, cases were demonstrated in which 40 to 50 dcb. above threshold hearing of different frequencies were required to mask the tinnitus, and instances in which tinnitus could not possibly be masked in any tried frequencies or masking noise.

In explanation, two kinds of tinnitus, vibratory and non-vibratory, were suggested — the first masked easily, the

second with difficulty. The ease of masking nonvibratory tinnitus depended upon the location of the lesion and the accompanying deafness.

The author, who has measured the loudness of tinnitus by the loudness balance since 1927, has described the technique. The tone or band of frequencies of the tinnitus was determined by the application of frequencies, first some distances above and below, then nearer and nearer to the patient's estimation until approximately the same tone or sound as the tinnitus had been obtained. If the tinnitus was above 8,000, it was often difficult to approach the frequency from the high tone side, from the uncertainty in judging high frequencies. Below 8,000 it was usually less difficult to judge even small frequency differences.

Both intensity and frequency of tinnitus should be determined.

In rating the diagnostic and prognostic values of the procedure the following points were suggested: if tinnitus was bilaterally similar in tone, coincident in timing and quality, it was probably central in origin. Monaural tinnitus might also be central.

If tinnitus or applied sound were bilaterally unequal in frequency or intensity, they appeared to the patient to be in one ear only because of the masking of the opposite ear. This might be substantiated by masking out the opposite ear.

Auditory hallucinations of tinnitus type might result from cerebral irritative phenomena and might arise from any physiological level, lesions in cochlea, cochlear nerve, ganglion cells, auditory tracts and protective pathways. These would not necessarily be central and might have little or no dependable localizing value in diagnosis.

It was observed by the author that tinnitus coincident with sharp dips or narrow deep troughs in the audiogram curve had its frequency in the limits of the slopes. He suggested the indication that both tinnitus and deafness arose from limited lesions in the basilar membrane, or in the associated spiral ganglia. It was improbable that the narrow gaps resembled or represented central lesions.

The tinnitus occurring during the irritative phase of the lesion foreshadowed subsequent degeneration of the neural elements involved.

Tinnitus occurring with a flat audiogram curve was less localized in frequency — was more like a roar, motor or waves. The author considered that tinnitus was of central origin in a large proportion of cases.

Tinnitus represented more than a subjective sound, rather a reaction of the neuromechanism occurring in certain extra-auditory and intra-auditory disturbances.

The author further commented that tinnitus mechanical in origin was easily masked by similar sounds slightly below it in frequency. Nonvibratory tinnitus (biochemical) would show variations in the masking, dependent upon the extent and position of the lesions which caused the tinnitus; even if it ceased, there might be little comparable change in the hearing, unless there was recovery in the neural mechanism involved. The more chronic the tinnitus the less probability there was of a cessation with probable gain in hearing. After further discussion of the masking effects on tinnitus, the author referred to the unfavorable outlook often given to patients with regard to head noises and the accompanying loss of hearing. Such might be avoided when one has at his command methods for the management of the medical and surgical treatment necessary to control the tinnitus. A cure for deafness should not be claimed just because the head noise disappears. Patients are apt to overestimate the hearing improvement when relieved of the annoying racket of noises.

The close relationship of head noises and deafness was often misunderstood. Deafness might occur without head noises but head noises could not occur without deafness. Many so-called "cures for deafness," even surgical, appeared to improve the hearing because of the relief from head noises.

In "Pathologic Changes in the Peripheral Auditory Mechanism Due to Avitaminoses (A, B Complex, C, D, and E)," Covell[2] stated that, unfortunately, except for beriberi, pellagra, scurvy and rickets, a clear-cut picture of a particular vitamin lack did not exist for the human. Our greatest need was to recognize physical signs which accompany mild or

moderate grades of dietary deficiency; the dietary prescription was difficult and confusing.

In previous articles the author had shown that experimental deficiencies in several vitamins produced degeneration of the cochlear nerve. This present report dealt with the histopathologic changes in the peripheral auditory mechanism, for avitaminosis, A, B_1, B_2, B_6, filtrate factor (antigray hair factor and chick antidermatitis factor), C, D and E. It was interesting in this connection to note the recent studies of Wohlbeck and Besse, that in vitamin A deficient animals, bone growth lagged behind that of the nervous system.

Little was known of the influence of vitamins on periods of prenatal development. A very careful analysis of all the animal studies was given and these were the general conclusions:

1. Vitamin A deficiency in rats was associated with middle ear infection, increase in the periosteal layer of the otic capsule, occasionally exostoses in the internal auditory canal, degeneration of the cells of the stria vascularis, external sulcus cells and parts of the organ of Corti, with slight to moderate demyelinization of the cochlear nerve.

2. Vitamin B and various factors of the B complex resulted in higher incidence of middle ear infection; degenerative changes were found in the external hair cells of riboflavin deficient animals, spiral ganglion cells were altered in appearance in all avitaminoses of the B group, less for B_6. According to the present series, the three most important vitamins for the maintenance of the myelin sheath of the nerve were in order, Vitamin B_6, Thiamin Chloride and Riboflavin. The cochlear was more susceptible to alterations than the vestibular portions of the VIIIth nerve.

3. Vitamin C deficient guinea pigs revealed hemorrhage into the lining of the middle ear cavity with an increase in thickness of the submucosa. Cells of the organ of Corti, including the hair cells, were swollen and degenerated, and also some of the cells of the stria vascularis.

4. Vitamin D deficient rats had infections of the middle ear associated with a laying down of osteoid with the ossicular chain and otic capsule. Demyelinization of the cochlear nerve also accompanied this vitamin lack.

5. Vitamin E low rats showed middle ear infection; fibrous tissue replacements in the muscle bundles of the midde ear; small exostoses and hemorrhage about the nerve of the modiolus occurred.

According to Shambaugh[3] in "Diplacusis. A Localizing Symptom of Disease in the Organ of Corti," diplacusis in deafness presented valuable pathologic diagnosis, as theoretically and clinically diplacusis could only be caused by a lesion of the organ of Corti. This, if true, suggested practical application in the diagnosis and treatment of certain types of deafness and Ménière's syndrome.

The author presented three varieties of diplacusis (the hearing of one sound as two):

1. Diplacusis binauralis echotica, in which one sound is heard a fraction of a second later than the other, as a distinct echo.

2. Diplacusis monauralis dysharmonica, a pure tone heard by the affected ear as a double tone of two pitches.

3. Diplacusis binauralis dysharmonica, same sound heard at different pitches by the two ears.

The diplacusis test was made with tuning forks and a complete octave series from 64 to 2,048 was employed. The author referred to the explanations of diplacusis offered by Politzer, Gradenigo, Jacobsen, Schilling and others. In 1935, the author described a syndrome of low tone nerve deafness, diplacusis and attacks of vertigo in substantiation of the theory that diplacusis could only be caused by distortion of the vibrating membrane of the organ of Corti.

An analysis of 45 patients was made, with 12 individual case reports.

The author presented his reasons for believing that the defect in perception of low tones was due purely to a lesion of the inner ear:

1. Perception as measured in the Schwabach test was always shortened.

2. When reaction to the Weber test was lateralized, the sound was always heard in the better hearing ear.

3. The Rinne test remained positive even with considerable defect in the affected ear.

4. The drum membrane and Eustachian tube were normal and inflation did not demonstrably improve hearing.

5. Marked fluctuations in hearing were not demonstrably due to the middle ear or Eustachian tube.

Chronic or recurring involvement of the membranous labyrinth presented this symptom complex or syndrome: Attacks of vertigo; inner ear deafness with marked loss of hearing for low as well as high tones, and marked fluctuations; diplacusis and tinnitus, both with tendency to fluctuate.

The author made the following conclusions: that *1.* diplacusis was due to a lesion of the organ of Corti; *2.* diplacusis was a more frequent symptom than usually supposed; *3.* the test for diplacusis by tuning fork was simple, quick and reliable; *4.* diplacusis occurred in otosclerosis with labyrinthine involvement, acute serous labyrinthitis secondary to suppurative otitis media, operative trauma in the membranous labyrinth, labyrinthine hemorrhage, labyrinthine acoustic trauma, labyrinthitis from local infection or allergy; *5.* diplacusis was found in most cases of Ménière's disease with defective hearing; *6.* Ménière's disease was due to nonsuppurative labyrinthitis, sometimes allergic and sometimes from focal infection; *7.* perception deafness, from lesion of the membranous labyrinth, showed impairment of hearing for low tones, presence of diplacusis, frequency of vertigo, reversible changes in the pathologic lesion, at least in the early stages.

In "The General Problem of Deafness in the Population," Beasley[4] presented a study of impaired hearing in the general population, applying to this problem the principles of epidemiological observation which have been applied to the general study of disease. He stated that impaired hearing was a functional morbidity, varying in degree and type with a wide range of etiology and predisposing factors, constitutional, environmental, and nutritional deficiencies, systemic and localized infections, endocrine abnormalities and toxic drugs. Knowledge of the rate of prevalence and incidence of impaired hearing gave basic information for significant trends. Present knowledge was limited to data from surveys of hospital patients and observations on army recruits.

Specific consideration was given to prevalence and annual incidence of severely impaired hearing in relation to age, sex, family income, employment status and occupational classes; and the prevalence of progressive high tone deafness involving minor and moderate impairment for conversational speech.

In the National Health Survey, enumerators inquired as to type of impairment classified in five groups: *1.* partial deafness, stage 1, difficulty in understanding church, theatre or group speech; *2.* partial deafness, stage 2, difficulty in hearing close range conversation but able to use telephone and understand loud spoken speech; *3.* partial deafness, stage 3, difficulty with the telephone but ability to hear amplified speech by hearing aid; *4.* total deafness for speech; *5.* deaf-mute.

The clinical investigations were carried out in temporary clinics in 12 different cities, where ear, nose and throat examinations were made and hearing tested by means of the Western Electric Audiometer. Both ears were tested by air conduction, and six tones by bone conduction. Audiograms on these averages were presented, and from the character of these losses it was inferred that hearing impairments were predominantly nerve deafness, which is contrary to the commonly held view.

In a study of annual increase rates of new deafness cases, it was apparent that all the morbidity had periods of acceleration and retardation in relation to age. By prevalence rates, the age period from $2\frac{1}{2}$ to 10 years appeared more serious than the whole period from 10 to 30 years. Rate of incidence rose rapidly from 30 to 40 years for both sexes. From 40 to 50 years, this continued to rise rapidly for females, and after 50 years the incidence rose again for males.

It was noted that for males and females of all ages, income exerted a relatively greater influence on the prevalence of impaired hearing among males than females. The highest prevalence of impaired hearing occurred among skilled and semi-skilled workers in industry; the second highest rates were among semi-skilled and unskilled workers; and the lowest among professional, business and clerical persons.

A higher prevalence of impaired hearing among the unemployed, reflected selection by employers in favor of employees who were not hard-of-hearing when large scale layoffs occurred. The author observed that unemployment prevalence was 60 per cent higher among persons with moderate deafness, and 98 per cent higher among those with severe deafness, as compared with the rates of those with good hearing.

After studying various sequences, conclusion was made that loss of good hearing with advancing age was balanced chiefly by increases in the prevalence of chronic progressive deafness; that moderate degree of high tone loss in one decade symptomatically indicated more progressive high tone loss in subsequent decades.

According to Bast,[5] in "Development of the Otic Capsule. V. Residual Cartilages and Defective Ossification and Their Relation to Otosclerotic Foci," the occurrence of residual cartilage or defective ossification in certain parts of the petrous bone in late fetal life or early childhood had been recognized for some time. In the region of the fissula ante fenestram, this defective development was found in a large percentage of fetuses and young children by Bast and his co-workers.

They suggested an unstable condition of the fissula ante fenestram as one of the factors which initiated a late attempt at rebuilding bone. Such residual cartilages occasionally occurred in the region of the fossula post fenestram. Otosclerotic bone had been observed in other regions of the otic capsule.

A review of the literature gave the impression of extension only from the fissula ante fenestram, but cases were recorded with multiple foci in various portions of the petrous bone which were not contiguous. Bruhl, in 1905, noted otosclerotic bone along the facial canal; others in region of the internal auditory meatus and base of the cochlea, and following the blood channels.

In summary, the author reported residual cartilage or infective bone in the following regions of the petrous bone: *1.* the region of the fissula ante fenestram; *2.* the region of the fossula post fenestram rarely; *3.* the infracochlear region; *4.* the

base of the styloid bone; *5.* the region of the petrosquamous suture, and in the capsule beneath the suture; *6.* the region of the semicircular canal.

Covell,[6] under the supervision of Prof. Nager in his laboratory, searched for the pathologic changes which resulted in immobility of the ossicular chain, observed any bony changes within the ossicles which might be correlated with the lesion in the labyrinthine capsule, and reported them in his article, "Ossicles in Otosclerosis."

Sections of 21 temporal bones which showed no otosclerosis and were classified as normal, were used as controls. Forty-six temporal bones (18 males and 14 females, age 20 to 88 years), which showed the otosclerotic lesion in the region of the stapediovestibular joint, were studied.

In a large percentage of the otosclerotic temporal bones, the sections showed changes in the architecture of the ossicles in the vicinity of the malleoincudal joint. These alterations resembled the so-called blue mantles of otosclerosis.

Typical otosclerotic foci occurred in the malleus and incus of both temporal bones of one individual. This was of interest because it represented the first time an otosclerotic lesion had been observed outside of the otic capsule. There were preotosclerotic conditions occurring, similar to those found in the labyrinthine capsule.

Covell believed that the observations of Holmgren on the diminished movements or even failure of excursions on the part of the shaft of the malleus were explained and should be of certain value in the diagnosis of otosclerosis.

Otosclerosis which resulted in fixation of the stapediovestibular joint impaired the mobility of the ossicular chain by displacement of the incudostapedial and malleoincudal joints. Even without ankylosis, a lesion in the oval window margin impaired the efficiency of the ossicles as a conducting mechanism.

Gardner and Turner[7] presented "Bilateral Acoustic Neurofibromas. Further Clinical and Pathological Data on Hereditary Deafness and Recklinghausen's Disease." Ten years ago, a history was reported of a family in which Recklinghausen's

disease in the form of bilateral acoustic tumors, had been transmitted as a dominant Mendelian trait through five generations. At that time the diagnosis was verified histologically in two cases, although the evidence indicated that 38 members were affected. The earlier descriptions of this family have been summarized and brought up to date. The gross and histologic observations in the cases of four additional affected members were described. One of the cases occurred in the sixth generation of this family. One individual had bilateral acoustic tumors 1.0 to 1.5 cm. in diameter — the smallest bilateral acoustic tumors described.

Considerable work has been done, showing that the form of Recklinghausen's disease which occurred in the central nervous system might be transmitted as a dominant Mendelian trait, often without the usual peripheral manifestations.

Weber,[8] in "Deafness Associated with Pigment Degeneration of the Retina," reported that much research had been in progress, to clarify the disease picture of deafness in combination with pigment degeneration of the retina.

Some prominent Vienna authors accepted a single causal injury as responsible for all hereditary diseases of the inner ears and referred to it as "acoustic hereditary degeneration." They claimed that two recessive genes caused otosclerosis. It was postulated that recessive deafness could show itself in many ways, from insignificant changes of the stria vascularis and aplasia of the organ of Corti and the nerve trunks, to serious changes in the cochlear nucleus and the brain itself.

The dominant form of hereditary labyrinthine deafness presented all stages, ranging from hard-of-hearing to complete deafness in the same family. In this type there were significant malformations of the cochlear and the peripheral nervous apparatus.

There was much controversy on the question of whether the coincidental occurrence of deafness and pigment degeneration of the retina was due to some inner causal connection or whether each was caused by independent existing anomalies. Histologic research of the brain in deafness and pigment degeneration of the retina which showed changes in the central auditory and optic tracts, resulted in findings which precluded the probability that these two illnesses had any common causal link.

He concluded with the statement that with so many and varied opinions on this problem, no single progressive step had been possible toward its solution.

In "Unilateral Hereditary Deafness," Smith[9] presented the case of a family in which four children out of eight had unilateral severe deafness without a history of ear infection. The mother of the children was "stonedeaf" in one ear also. The vestibular function by caloric tests was normal in all of them. The children who resembled the mother were deaf in one ear; those resembling the father had normal hearing. In tracing the family, it was found that in three generations of the descendants of one man with unilateral deafness, there were nine persons with hereditary deafness.

Geshelin[10] wrote "On the Pathogenetic Structure of Otosclerosis." Basing his observation on 35 cases of otosclerosis, he emphatically denied any pathogenetic connection between otosclerosis and the endocrine system.

In "Injury to Tympanic Membrane Caused by Explosions," Craig[11] described six cases of the effect of these injuries in the present war. None of the cases had worn any protectors and none received any treatment until they arrived in the hospital one to nine days after the injury. In all the injury was to the tympanic membrane; most developed acute otitis media; hearing loss varied; no case suffered from vertigo. All cases were treated by clearing away discharges, blowing in iodine and boric acid, or dressing with one-half inch ribbon gauze soaked in acriflavine 1-1,000, and all were given sulfanilamide. The author considered protection to the ears essential and felt that no type of plug had any advantage over cotton wool impregnated with vaseline, soap or candle grease.

Under the title, "Usable Hearing," Bunch[12] stated that total loss of hearing, either bilateral or unilateral, occurred with relative infrequency. Considerable residual hearing might be found in audiometer tests with proper masking. Sometimes patients were even able to hear and understand loud conversation or words shouted close to the ear, or at least the sound of the shouted voice. Similarly, it was reported that 95 per cent of the children in the Pennsylvania School for the Deaf responded to at least one tone in one ear by air conduction.

The usefulness of such residual hearing depended upon its definition. Even loud conversation at close range could be, in a certain way, usable.

From the Fletcher experiments it was stated, "when the intensity of a tone is continually increased, a value is reached where the ear experiences a tickling sensation." Pain occurred above this feeling point. Consequently, the feeling sensation was maximum intensity to be useful for practical hearing.

The studies in the telephone laboratories to obtain curves representing total loss of serviceable hearing have proved difficult, though the curve based on the theorem, "total deafness for speech occurs for an average hearing loss of 85 dcb.," had a certain amount of correlation value.

The author in his summary stated that evidence was presented "for correlation between audiometric tests and tests with shouted voice in cases of profound deafness. On the basis of this correlation, a curve was proposed which furnished in audiometric tests, a dividing line between usable hearing and that which was not usable."

After quoting opinions from three noted otologists, Macfarlan,[13] in "Speech Hearing and Speech Interpretation Tests," discussed important points in the testing of speech hearing. Heretofore, audiometry had principally meant the determination of pitch hearing, with little or no correspondence between the hearing for the various pitches and that for speech. There had been a growing conviction that the only way one could determine speech hearing was by using speech as the testing stimulus. With a growing consciousness that the deafened were most interested in how well they could hear the human voice, it had become necessary for the otologist to have a thorough knowledge of speech hearing.

The author has given a careful outline of procedure in the study of the hearing of speech, which included the composition of language, characteristics of spoken language, and reasons for hearing the spoken voice poorly.

The English language has proved particularly difficult in three respects: *1.* words were not pronounced as they were spelled; *2.* words with different spelling might have the same pronunciation; *3.* words with the same spelling were pro-

nounced differently. In discussing the significance of the phonetics of speech to the otologist, the author stated that speech represented an infinite variety of sounds and frequencies. Sounds were noisy, musical, explosive, sibilant, resonant, aspirated, nasal, soft, hard, loud, thin or rich in tone color. Each sound pattern had a meaning or significance. For each there was a memory and an association.

Speech hearing could only be tested by the use of speech as a test stimulus. The motor-driven phonograph, with its electrical pick-up and decibel meter, has revolutionized testing for speech hearing. Any desired intensity could be obtained and repeated with identity. As the word list should be selected with great care as to articulation, quality of recording, needle scratch and word familiarity, the author presented useful lists of words.

After discussing the otologist's help in choosing a hearing aid, determining whether hearing was improved by suppressing low or high tones, testing the speech hearing of very young children, and judging of central perception, the author answered as in the form of a questionnaire important natural queries.

In conclusion, it was stated that speech hearing tests must be approached with an understanding of the characteristics of speech. The patient was primarily interested in speech hearing; there was a correlation between speech hearing and frequency hearing; speech hearing must be tested by the use of speech; the phonograph should be used in place of the old spoken voice test, and a knowledge of speech hearing tests and the proper equipment should be in the armamentarium of every otologist.

In answer to the query, "Is Improved Hearing Possible in Patients Who Have Residual Auditory Perception?", Baldrian[14] stated that the natural courses of learning the spoken word, were closed to congenitally deaf children, as well as to those who had been deaf since earliest childhood. Schools have been established with the purpose of making the handicap an easier one to bear.

The author pointed out that through long and tedious tests, the hearing ability of both ears could be determined and with

the proper approach the shape of speech followed slowly but surely, and with the development of intellectual and social senses the whole psychology and morale of the patient was raised. The merging of auditory and visual impressions in combination with the psychological effect aided the pupil in a better understanding.

He stated there was no literal improvement in hearing in the case of those subjects with residual hearing, but the improvement was rather in the fact that by the development of the ability to sense tones, the need of hearing speech was minimized.

"Estimation of Improvement in Hearing Following Therapy of Deafness" was discussed by Hughson and Witting.[15] They stated that for many years in the prevention and alleviation of deafness the prompt recognition and immediate therapeutic measures in upper respiratory and ear infection, had been the chief concern. The vitamin and endocrine theories and Crowe and Baylor's assertion in regard to adenoid growth at the Eustachian orifices, have absorbed much attention.

In a survey of 313 families of seriously deafened children, hereditary deafness was established in only 5 per cent. In only 16 families were both parents deaf. In 13 instances the father alone was deaf; in 11, the mother alone. The authors discussed the reliability of present standards of measurement and felt that soundproof examination room, proper masking and establishment of the relation between bone conduction and nerve deafness should be considered. With a definite difference in threshold, the loudness balance test was important for differential diagnosis, though often unavailable. When the results of such loudness balance and bone conduction findings conformed to each other, an absolute diagnosis of either conductive or nerve deafness could properly be made.

Certain standards of measurement were suggested and the authors believed that medical and surgical enthusiasm for newer forms of treatment required rigid analysis and a common standard of interpretation for end-results.

Frequently repeated audiograms were indispensable. In the consideration of rehabilitation, losses of 50 dcb. or more made

the achievement of really useful hearing uncertain. The arbitrary figure of 10 dcb. has been chosen as the minimum improvement to be considered of real consequence, and necessary duration of improvement has been set at five years.

Kobrak, Lindsay and Perlman,[16] in "The Next Step in Auditory Research," stated that in the past the anatomic structures of the middle ear have been studied intensely. Each part of the cochlea had been diligently described and carefully measured. The function of each part had not, however, been studied satisfactorily. The students of hearing theories, therefore, had based their assumptions mostly on structural and not on functional considerations. This was, unfortunately, one-sided and might be the reason that several auditory phenomena could not be explained on the present anatomic basis. More physiologic experimentation on the function of the structures of the inner ear seemed to be the most desirable "next step in auditory research."

The intralabyrinthine fluid was an essential conductor of acoustic energy. Disturbance of the cochlear fluid was incompatible with good function. Direct conveyance of acoustic energy through the bone to the nerve endings was not possible. Regardless of the portal through which the sound entered the cochlea, the intralabyrinthine fluid was the final common path of sound conduction.

According to Fischer,[17] in "Changes in the Internal Ear Due to Increased Endocranial Pressure," it has not been definitely determined whether changes in the inner ear occurred similarly to choked disc in the eye as a result of increased endocranial pressure. The author has attempted to clarify these conditions by histologic study, and he has discussed in detail the microscopic examination of serial sections of petrous bones, particularly occurring in chronic increase of endocranial pressure.

The following conclusions were drawn:

1. The histologic changes in generalized brain pressure followed six main groups:

a. Transudation, exudation and lymph congestion in the region of the membranous inner ear and the nerve ganglion apparatus.

b. Hyperemia, hemorrhage and changes in the blood vessels of the membranous inner ear and the petrous bone.

c. Formative changes (ectrasia, stenosis and collapse) of the ductus cochlearis, the ductus reuniens, and the saccus and ductus endolymphaticus, and perilymphaticus.

d. Multiple hernias of the brain, especially on the anterior surface of the pyramids and in the middle fossa, accompanied by pressure erosion of the underlying bone.

e. Secondary (late) changes, pigmentation, adhesions and degenerative atrophic changes of the sense organ and the nerve ganglion apparatus.

f. Destructive osseous changes (pressure atrophy).

2. The pathways by which the brain pressure was transmitted into the inner ear were the subarachnoid spaces of the aqueductus cochleae, the nerve sheaths in the internal auditory meatus, the Pacchionian granulations, the spiral ligament, the lymph channels of the bony labyrinth, and the blood vessels.

3. An analogy between choked disc and congestive inner ear was justified.

4. Increased generalized brain pressure may have existed over a long period, producing secondary degenerative changes.

Because of the paucity of reports in American literature. Hagens,[18] in "Pathology of the Inner Ear in a Case of Deafness from Epidemic Cerebrospinal Meningitis," presented a brief case history and discussed the postmortem examination of the temporal bones. Grossly there were no signs of infection in either ear.

The author presented a most minute and detailed description of the labyrinth changes in each ear, more marked on the right side.

In summary, there was apparently in the beginning a diffuse suppuration of the labyrinth; at the time of death, 53 days after, the process had undergone resolution and considerable replacement by granulation tissue. Bone destruction was found about the semicircular canals but there was only moderate evidence of bone rebuilding. In the right ear, the

spiral ganglion, organ of Corti and vestibular organs were practically destroyed, though Scarpa's ganglia was intact. In the left ear, spiral ganglion was present and a distorted organ of Corti remained in the basal coil.

Both divisions of the VIIIth nerve were present in the internal auditory meatus, but polymorphonuclear leukocytes along the nerve tissues indicated the pathway of spread of infection into the labyrinth. The fossae for round and oval windows, and pneumatic spaces, showed moderate infection but no evidence of pathway from these to the labyrinth.

The author then reviewed Politzer's valuable discussion on this subject, who stated that the deafness arising in the course of a primary form of meningitis was evidenced at return of consciousness, between third and eighth week of the disease, or developed during convalescence. Here, too, anatomic and pathologic changes were noted in minute detail. According to Politzer, deafness which remained after epidemic cerebral meningitis was usually bilateral — disturbances of equilibrium were noted in more than two-thirds of the cases.

In reviewing the survey of schools for the deaf, with examination of 5,348 children in 1930, 40 per cent were judged to have acquired their deafness, and of these, 385 apparently followed epidemic cerebrospinal meningitis. The result showed that epidemic meningitis tended to produce severe or total deafness with marked or complete loss of the vestibular function.

In final conclusion, the author noted that deafness occurred 46 days before death and that the findings supported the reports in European literature of a severe destructive bilateral labyrinthitis with tendency toward healing, showed by granulation tissue in bone. The end-organs were practically destroyed, demonstrating the reason for loss of cochlear and vestibular function.

Hardy[19] presented, under the title, "Deafness and Retinitis Pigmentosa," the case of a boy, age 11 years, who had deafness and an almost complete loss of retinal pigment. It was noted about midway to the periphery, that the retina was dotted with fine pigment, giving a stippled appearance. The

audiogram showed a progressive and steep loss as one passed from the low tones through the middle to the high tones. Two other cases in older individuals were mentioned who showed similar audiometric changes, and both had retinitis pigmentosa.

In a careful review of the literature, Simonton[20] has analyzed the meaning of "Ménière's symptom complex." Ménière, in 1861, described the triad of symptoms of vertigo, tinnitus and nerve deafness as a clinical entity. Early writers applied the term Ménière's disease or aural vertigo to all ear conditions where vertigo was a symptom. Later, the term Ménière's disease, now more preferably termed Ménière's symptom complex, was isolated to that group of cases without etiologic background, in distinction from those of special pathologic relations.

Ménière's report was classic, noting the sudden onset of vertigo without preceding illness, recurrence of attacks, tendency of spontaneous remission of the symptoms, the extreme discomfort of the patient during the attack, and increase of vertigo with movement. He observed that tinnitus preceded the first attack, persisted between attacks, often increased during the attack, and was not affected by pressure on the carotid artery. Impaired hearing was usually in one, though sometimes in both ears, began with tinnitus and progressed to complete deafness without history or findings suggestive of ear disease.

The etiology of cerebral vascular accident was opposed by the following: *1.* paralysis did not occur; *2.* patient remained conscious; *3.* recovery occurred rapidly; *4.* attacks were recurrent; *5.* neurologic findings were normal; and *6.* loss of memory was not associated with the attacks.

In his review the author cited the 11 causes of vertigo given by Knapp in 1871; the classification of aural vertigo according to peripheral, blood pressure and general systemic causes by Lake in 1912; the review of recent literature by Monroe in 1937, suggesting 13 definite causes of Ménière's symptom complex; the medical and surgical therapeutic review by Ramadier in 1933, and many individual references of importance in this and other countries.

In conclusion, the author quoted the review of Walsh and Adson on 182 cases of Ménière's symptom complex treated at the Mayo Clinic:

1. Spontaneous recovery was doubtful, although remissions frequently occurred.

2. Complete deafness or loss of vestibular reactions on the affected side was no guarantee for disappearance of vertigo.

3. Medical treatment by modification of Furstenberg regimen was of definite value.

4. Total or subtotal intracranial section of the acoustic nerve was the treatment of choice for patients not relieved by medical management.

In "Syphilis as Seen by the Aural Surgeon," Rodger[21] reviewed his experiences over a 10-year period in which he saw 500 cases of ear, nose and throat syphilis. In regard to congenital syphilis he stated that we are only concerned with the secondary and tertiary stages of the disease. There was an early and a late type. In the early type there was frequently an otolabyrinthitis affecting the labyrinth and middle ear. The late type was exemplified by deafness following interstitial keratitis, generally about the age of 9 years, and this was mainly labyrinthitis. The lesions of the ear were ultimately bilateral, started on one side, and weeks or months later the other was affected. In the early cases the destruction of hearing occurred before speech was acquired and the patient was destined to be a deafmute, and in the late type, speech was only altered to the monotonous voice peculiar to the very deaf.

He discussed the early cases and stated that the first lesion was a meningoneuritis affecting the VIIIth nerve. Secondly, there was an attack on the middle ear and labyrinth. There was sudden onset without pain or malaise, and a little pus from the ear.

The most distressing reflection in connection with these cases of the early type was the fact that nearly always the diagnosis was made in retrospect, after the internal ear was damaged beyond repair.

In discussing deafness of later onset in congenital syphilis, he stated that in his series it occurred in 44 out of 121 con-

genital cases. In this type there was an involvement of the labyrinth without involvement of the middle ear. It came on suddenly without the other signs of otitis and might start as early as 4 years and as late as 45 to 50 years of age. The deafness was profound and came two or three years after the keratitis. This form of deafness probably never occurred in acquired syphilis and was most common in childhood or adolescence. It was twice as common in females as males. Tinnitus was a very frequent symptom. Vertigo was almost as frequent as tinnitus.

In acquired syphilis of the ear, the most common condition in the secondary and tertiary stages was a neurolabyrinthitis without middle ear involvement, and was usually bilateral. The author has seldom seen any improvement in hearing after prolonged medication.

Witting and Hughson,[22] under the title, "Inherent Accuracy of a Series of Repeated Clinical Audiograms," reported on investigations on a long series of audiograms taken to obtain a quantitative determination of errors in hearing measurements on hard-of-hearing patients, and the probable relationship of errors on the hard-of-hearing and normal individuals; 297 audiograms were made in this investigation on 17 patients.

In their conclusions they found the hard-of-hearing individuals exhibited an inherent error of slightly less than 5 dcb. If the minimum error at 1,024 cycles can be considered indicative of the maximum ease of listening, the procedure of using this as a starting point in an audiogram might be considered justified.

Large occasional deviations from the average were experienced and were unexplained. Evidently these large deviations were to be expected in hard-of-hearing patients, but large deviations occurring too frequently should not remain unexplained.

The lowest frequency used, 128 cycles, showed such remarkable correlation to 256 cycles that the audiogram might justifiably be terminated at that point. Variations greater than those obtained in this study were assumed to arise from such contributing factors as tinnitus, therapy or infection.

In "VIIIth Nerve and Conduction Deafness," Selfridge[23] stated that sufficient evidence had already been offered to show that there were many diseases involving the general nervous system and the peripheral nerves, the etiology of which in the past was unknown but which were now definitely recognized as being related to nutritional deficiencies. The auditory nerve was a peripheral nerve — one of the most vulnerable in the body, and it should, therefore, show an earlier and greater damage than peripheral nerves elsewhere.

A recent survey of 9,000 individuals in 12 American cities by the United States Public Health Service showed that 50 per cent were hard-of-hearing in varying degrees, and that a large number of children below the age of 10 years showed beginning loss at the upper end of the tone scale.

Most deafened individuals did not understand that there was a definite difference between deafness due to degeneration of the nerve and internal ear and conductive deafness due to changes in the middle ear, drum or ossicles.

The author referred to the experiments of research workers in vitamin deficiency and pointed out that there were several factors in the vitamin B complex and vitamin E which were being studied in relation to nerve changes. He also mentioned Crowe's work with radium to destroy the lymphoid tissue blocking the Eustachian tube mouths and stated that in addition he corrected the patient's dietary and environmental factors.

Most of the author's work was based on 24-hour urinary output of thiamin chloride and porphyrin. In nearly 100 per cent of the cases, vitamin B_1 output was below normal, and approximately 50 per cent of the cases showed increased porphyrin — the latter substance has been found to be universally present in pellagra, hence proof that the use of nicotinic acid was indicated. His recent practice was to prescribe thiamin chloride (1,000 I.U.), nicotinic acid (gr. 1) and milk sugar q.s. as grs. V per capsule, one capsule after breakfast and after dinner. He supplied necessary endocrine gland products to those deficient either in pituitary, thyroid or sex hormones. If the blood count and hemoglobin were not normal, the proper therapy was prescribed. Studies of creatinin and creatin in the urine were made and if deficiencies were

present, indicating muscular dystrophy possibly of the stapedius and tensor tympani, glycine and prostigmin were given to overcome this disorder.

He stated that there were varied views on otosclerosis and that it was extremely difficult to make the diagnosis with certainty during life. Some authors claimed that 70 per cent of deaf people had otosclerosis; others said not over 6 per cent. Environmental factors were believed at present to exert more influence than hereditary factors.

He commented on an article by Nash, who attempted to restore normalcy to the conductive mechanism in the middle ear by various measures, including inflation of the Eustachian tube, otomassage, correcting nasopharyngeal pathology, removal of tonsils and adenoids, thermal procedures, endocrine therapy and vitamins B and C, and stated that many of the things mentioned were definitely related to faulty diets plus other metabolic disturbances.

Selfridge stated that he believed the time to begin treatment of a prospective otosclerotic was before birth. The mother should have an optimum diet during her entire pregnancy, and the child treated the same way during infancy, childhood and adolescence.

He pointed out that the important dietary factors were one egg daily, one pint of milk daily, one leafy salad daily, orange, grapefruit or lemon daily. Wittmaack and others made extensive studies of the injurious effects on hearing, caused by noise in industry. They recognized, however, that dust, changes in temperature, physical exertion and chemical influences played an equally harmful rôle.

Jankoff[24] reported on "Effect of Temperature Fluctuations on Hearing." Temkin, who had done research on the effect of temperature changes in relation to occupational hearing impairment, believed that the various factors mentioned caused injury to the upper air passages and extended through the Eustachian tube into the middle ear, and produced atrophic changes in the labyrinth following the middle ear catarrh. No one has confirmed these findings. Consensus of opinion was that temperature changes affected the labyrinth directly.

The author studied hearing impairment in industrial workers and compared the results with those obtained in studies of locomotive drivers and stokers afflicted with hearing impairment. He found that the percentage of deafness was higher among the locomotive drivers. He further found in his studies on 310 locomotive drivers that the hearing in the right ear was always less than in the left. He explained this by stating that the right ear of the locomotive driver was subject to more frequent and much more severe changes in temperature than the left.

He stated that occupational deafness presented a series of problems which awaited solution. He suggested the following factors: *1.* better regulation of working hours; *2.* specified rest periods; *3.* adequate ways of protecting the worker against severe temperature changes, excessive noise, dust, etc.

In "Practical Questions on Pathology of Heredity in Otiatrics," Kecht[25] stated that the question of whether or not hereditary influences were generally indicated in congenital deafness, was one of first importance. There were two views on the causes of hereditary congenital deafness. First was that of Hammerschlag, who believed that all types of impaired hearing and deafness were caused by a single noxa, and manifested the different forms according to the degree of illness. The second view, held by the majority of otologists of The Reich, was opposed to the Hammerschlag view, and contended that various types of hereditary deafness could be traced to different injuries. They classified the hereditary types as follows:

1. Congenital Degenerative Inner Ear Deafness. There was defective development of the cochlea, organ of Corti, and defective nerve structure was often seen. The disease manifested itself in early youth. Clinically, there was greatly reduced bone conduction, and in such cases sterilization of the carrier was indicated.

2. Sporadic or Recessive Deafness. This occurred only as total deafness and anatomically it involved the nerves. There was faulty development of the auditory nerves and defective central nuclei. In approximately 10 per cent of cases in this form of deafness there occurred associated pigment degeneration of the retina. Sterilization of the carrier was suggested.

3. Congenital Auricular Atresia. This malformation could be divided into three genetically similar groups: *a.* Defective tympanic ring and rudimentary membrane. *b.* Serious malformation of the tympanum. *c.* Tympanic cavities undeveloped.

4. Otosclerosis. In this disease there was defective construction of the bony labyrinth capsule. The normal bone structure underwent resorption and new and plexiform bones were formed. Many well known authors were opposed to sterilization laws in cases of otosclerosis. Albrecht believed that sterilization should be voluntary.

Forbes[26] stated that a number of drugs and chemicals have a predilection for the VIIIth nerve and were capable of causing nerve deafness. The list includcd quinine, salicylates and alcohol in particular; also tobacco, arsenic, lead, phosphorus, carbon monoxide, carbon disulphide, oil of chenopodium, mercury, morphine and the aniline dyes. In "The Etiology of Nerve Deafness with Particular Reference to Quinine" the author claimed that quinine caused more deafness than any other. Its most frequent use was in the treatment of malaria, but in addition it was used as an abortifacient, an oxytocic, in the treatment of influenza, diseases of the upper and lower respiratory tract, Ménière's disease, corneal ulcers, tic douloureux, cardiac diseases, etc.

Information from all available sources indicated that the consumption of quinine had been as great in the decade immediately passed as in the one preceding. There was evidence, both experimental and clinical, which supported the view, that quinine administered to the mother during pregnancy might cause deafness and amblyopia in the child. In some instances it might cause both hearing impairment and reduce visual acuity when administered in large doses to a child.

In a series of 234 cases of nerve deafness and 832 cases of deafness other than nerve deafness, the percentage of patients who had a history of taking quinine was four times greater among those with nerve deafness.

Kelly[27] stated that "Deafness and Loss of Vestibular Function Following Herpes of the Scalp" was comparatively rare. This condition, known as herpesoticus, had three principal varieties:

1. Herpes of the external auditory meatus associated with facial palsy due to involvement of the geniculate ganglion.

2. Herpes with facial paralysis and deafness (geniculate and spiral ganglion).

3. Herpes with facial paralysis and vestibular disturbance (geniculate and Scarpa's ganglia).

The facial palsy recovered unless the spiral ganglion or Scarpa's ganglion was affected, in which case paralysis and deafness or loss of vestibular function were permanent.

The case presented conformed to none of the types. The patient had a left-sided deafness for seven months. He had an attack of herpes at the time his deafness started and it involved the scalp on his left side — not the external ear at any time. There was no facial paralysis. He had some dizziness and a tendency to deviate to the left in walking with his eyes closed. The left side on examination was completely deaf. The author felt that the ganglia involved in this case were: *a.* The semilunar ganglion of the Vth nerve. *b.* The posterior root ganglia of the IInd and IIIrd cervical nerves. *c.* The ganglia of the VIIIth nerve — the spiral ganglion and Scarpa's ganglion.

He also discussed the connections between the Vth and VIIIth nerves.

The most important observation in the "Histologic Studies of the Eustachian Tube of Individuals with Good Hearing," by Polvogt, Leroy and Babb,[28] was the finding of lymphoid tissue and lymphoid nodules in the Eustachian tubes of people with normal hearing. They found that the vascularity of the tunica propria was usually greater in children than in adults. The tube was lined with ciliated epithelium, and it might be a straight, smooth-walled structure, but more commonly had a corrugated appearance, due to evaginations of the tunica propria and epithelium into the lumen.

The tube and bony portion might be like a cylinder with little surrounding pneumatization, or there might be extensive surrounding pneumatization. In these cases, dilatation of the tube with a bougie was difficult and hazardous, and these tubal cells afforded a pathway for the spread of infection throughout the temporal bone.

The part the lymphoid tissue and lymphoid nodules, which had been found even in late adult life, played in the impairment of hearing, these authors had not yet been able to observe, as this study concerned those with normal hearing.

In his superb review of recent literature on "Intradural Conditions in Relation to Rhinology and Otology," Eagleton[29] has abstracted the investigations of Dederding and Jerlang as to the postoperative auditory and vestibular symptoms of patients who, suffering with typical trigeminus neuralgia, had been operated upon by: *a.* Section of the sensory root with *b.* electrocoagulation of the middle meningeal artery, *c.* severance of the large and small superficial petrosal nerves to "prevent traction on the geniculate ganglia and facial paralysis."

Wilson[30] reviewed "Swift's Deafness, and His Last Illness" from other authors' writings and from Swift's diary. Otosclerosis, syphilitic labyrinthitis and other diseases had been mentioned as the cause of his malady, but the author concluded that Swift's deafness and attacks of dizziness were due to Ménière's disease.

In "Occurrence and Pathology of Otosclerosis," Engstrom[31] stated that interest in otosclerosis dated back several centuries, but starting in 1910, otologists recognized greater significance in this disease. The best known of the basic monographs on the clinical and pathologic anatomy of otosclerosis was by Wittmaack, who described the disease as congestive processes. He stated that in spite of world-wide research, a clear conception of these processes, especially as to etiology and treatment, was still lacking.

The author described his own histologic methods and results obtained. In only a part of his cases could there be a proper evaluation of the circumstances since there was no clear idea as to the frequency of the changes. Clear-cut otosclerotic changes were not very prominent.

He described surgical treatment of the disease and stated that the operation was a delicate one and that the operator should not be too confident of success the first time.

With proper ventilation through the Eustachian tube, Campbell and Hargreaves[32] believed that the only condition

to be dealt with in "Aviation Deafness — Acute and Chronic" was fatigue of the end-organ of hearing, which became chronic because of accumulated fatigue through severity and length of time. This in its severest form presented the usual conception of traumatic perception deafness. On the other hand, both closure of the Eustachian tube or its physiological failure in opening, as in sleep, failed to complete ventilation during the positive and negative changes in pressure produced by variation in altitude. The picture of conduction deafness might be superimposed in degree, depending upon the severity and duration of the application of pressure. This would explain the variation in audiograms which have fully illustrated the article.

The authors discussed fatigue in its general physiologic conception, the force of the fatiguing elements and the characteristics of the fatiguing force. Ultimately, through proper ventilation in soundproof cabins, there would not be a serious problem in flying.

Deafness of aviators might be classified as to cause:

1. Acute fatigue of the end-organs of hearing and related structures.

2. Chronic accumulative fatigue of the end-organ and related structures.

3. Changes in middle ear pressure occurring in changes in altitude, with faulty ventilation of the middle ear.

4. Chronic conduction deafness due to alteration in tissue resulting from faulty ventilation of the middle ear.

The first two groups had a decrease in the acuity of hearing near the 4,096 area of perception deafness; the last two a decrease for the lower frequencies, 128 to 1,024, a form of conduction deafness. Conduction and perception deafness might be combined to give any form of audiogram.

Fatigue of the end-organ of hearing was due to noise and other vibratory energies inherent with flight. The 4,096 area was particularly vulnerable because of exposed position and possibly altered blood supply. Additional decrease in oxygen might be a factor.

In "Gun Fire Deafness," Passe[33] reported that since the outbreak of the war many naval personnel have complained of deafness immediately following gunfire and bomb explosions. It occurred in men of any age and without reference to upper respiratory trouble in the past or present, and most cases were unilateral. The type of gun responsible for the majority of cases was the smaller type of 3-inch, 4-inch, 4.7-inch and salute guns. Deafness only occurred in those outside the protective shield of the enclosed turrets. The ear nearer to the mouth of the gun barrel was the one most frequently affected. Most cases had worn cotton ear plugs loosely; no case wearing rubber ear plugs had been reported.

Majority of gun crews were in a brief state of "auditory daze" after action, which lasted from seconds to mintues. High-pitched ringing sound was present during this period. If he put his finger in his ear, the patient might find it blood-stained. When the ship returned to port, the otologist consulted might find one of four conditions:

1. More or less rapidly recovering nerve deafness with a normal drumhead.

2. Nerve deafness recovering slowly and a raised darkish red area on the surface of the drumhead extending backwards from the umbo.

3. A nerve deafness with a ragged linear tear of the drumhead extending posteriorly, with blood in the canal.

4. A nerve deafness associated with a perforation, as in No. 3, plus middle ear infection. In every case, this followed syringing.

The loss of hearing over the whole range was very marked for the first 24 to 48 hours and gradually the hearing returned, with the subsidence of the tinnitus. The upper register was the last to return. Most cases did not recover their hearing completely. The author emphasized the use of rubber ear plugs and advised against syringing the ears following rupture of the tympanic membrane.

Davis[34] felt that "Deafness Due to Gun-Fire" might arise from injuries to:

1. The auricle and external meatus.

2. The membrana tympani and middle ear.

3. The internal ear.

When the auricle is torn off by a shell or the external canal injured, it was important to do a plastic repair so that sound could still get through. The commonest site for stenosis was the junction of the cartilaginous and bony meatus. Foreign bodies were more easily removed by postauricular incision.

The tympanic membrane was frequently ruptured following an explosion in a confined space, such as a trench. The perforation usually occured in the lower half of the drumhead and the patient complained of pain, deafness, bleeding from the ear, and vertigo. Obturators of various characters had been used to advantage to prevent this accident, but none was quite as satisfactory as the index finger plugged in the canal.

The most important treatment for this condition was rest in bed for a few days. All interference should be avoided. The drumhead would usually heal without loss of hearing in five to 10 weeks.

There were three clinical types of injury to the internal ear which arose from gunfire: *a.* Those with structural and permanent damage to the internal ear. *b.* Psychic or functional deafness. *c.* Temporary deafness from concussion.

In the first group the exact nature of the injury to the internal ear was not known, but Fraser, who made microscopic examination of the ears from four fatal cases of this kind, found hemorrhage at the fundus of the internal auditory meatus. Re-education and rest were the best treatments for the other two types.

Fox and Sweet[35] reported on the syndrome of "Brittle Bones Associated with Deafness and Blue Scleras" in two persons of one family. This syndrome of brittle bones, blue scleras, relaxed ligaments and deafness was recognized as an interesting hereditary condition, because of the nature of the hearing impairment and similarity to deafness in otosclerosis.

These patients, usually first seen in the orthopedic service because of frequent and multiple fractures, were primarily recognized by the blue scleras and the familial fracture history. The associated deafness in reports of brittle bones and

blue sclera were first made by Van der Hoeve and deKleyn, and later by Bronson.

This article concerned the history and findings in two patients, a brother and sister, with blue scleras, definite hearing impairment and multiple fractures. Characteristics of the fractures were: *1.* ease of occurrence; *2.* absence of pain; *3.* absence of predilection site; *4.* promptness with which healing took place.

It has been stated that among adults with blue scleras, 60 per cent have fragile bones, 60 per cent associated deafness, and 44 per cent all three defects. In the cases reported, both nerve and conduction deafness were present, with marked loss for upper tones, especially above the 4,096 cycles. According to the authors, the blue scleras and brittle bones were noticeable early in life; the otosclerotic type of deafness usually appeared in the late twenties.

In "Defective Hearing and Chronic Ear Disease as Revealed by the Examination of 47,000 Philadelphia School Children," Cornell[36] stated that since 1929 the Division of Medical Inspection of the Philadelphia Public Schools had tested the hearing of approximately 6,000 children each year with an audiometer which had multiple head phones testing one ear of 40 children at one time. The phonograph record reproduced a human voice which dictated numbers which the children wrote down on test sheets. The voice gradually became fainter. Those children who were defective in hearing to the extent of 30 sensation units could not hear any of the dictated words. Those with only a small loss wrote down the numbers first dictated, but were compelled to stop before the final faintly spoken ones were reached. The children tested were in the fourth school grade, averaging 10 years of age.

After the first test it was found that 9.3 per cent of the children had a definite hearing loss in one or both ears, and after retesting the defective ones, only 4.1 per cent of the children showed a definite loss, and after a third test only 3 per cent showed a definite loss. This change was accounted for by the fact that the children understood better what was expected, they were less nervous and there probably was a better adjustment of the head phones. The author stated:

a. Loudness of sound did not increase proportionately with increase in the amplitude of the air vibrations.

b. The creation of air waves which were identical with those already in existence did not double the amount of sound thereafter produced.

c. Hearing loss might be stated in percentage figures by assuming that six sensation units equaled 5 per cent.

d. If we took the pitch of middle C or of high C, hearing might be considered good if loss in hearing did not exceed 10 to 12 dcb.

Of the children with defective hearing (3 per cent), 1 per cent had defect in both ears, and 2 per cent in one ear only. One-half of the deafened ears showed damaged drumheads.

Panneton[37] wrote on "Functional Ear Disturbances of Tubal Origin" and restated the well known fact that deficient aeration of the middle ear led eventually to tinnitus, a sensation of fullness and progressive deafness. He stated that adequate physiologic ventilation depended upon an intact drumhead and ossicles, as well as an intact nose, nasopharynx and muscles of the palate.

He discussed in detail the examination of the tympanic membrane and stated that, regardless of the appearance, if it reacted to the movement of deglutition, one might exclude the possibility of inadequate aeration of the cavity. His experience had shown that if the membrane was entirely transparent or partially distended, the prognosis was poor from the standpoint of tubal therapy. Even if the membrane was perforated, he had found that functional disturbances might be the result of a tubal lesion. Every case should have a careful examination of the tubal orifice with the nasopharyngoscope, and a catheterization of the tube.

He felt that unilateral nasal obstruction did not prevent normal ventilation of the middle ear.

In children, most functional disturbances of the ear could be traced to adenoids, inadequate nasal respiration and tonsillar hypertrophy.

In adults, the most frequent causes were: chronic tonsillitis, hypertrophied ends of the inferior turbinates, atrophy

of the rhinopharyngeal mucosa, hypertrophy of the mucosa with lymphatic granulations about the orifice or within the tube, whitish edema of the anterior lip of the tubal orifice, and paresis or atony of the soft palate with scars following tonsillectomy.

He suggested for treatment: tonsillectomy, genital gland extract for mucosal atrophy, thyroid extract for mucosal hypertrophy, silver nitrate treatment of granulations and treatment of the tube with bougies carrying cotton dipped in silver nitrate.

He felt that the Politzer bulb and the Itard sound might cause damage, but if used properly these instruments of treatment were valuable.

Moore[38] has ably discussed "Neurologic Problems Past 50," under five headings:

1. Vascular disorders.
2. Intracranial space-taking lesions.
3. Degenerative diseases.
4. Inflammatory diseases.
5. Miscellaneous conditions.

The many subdivisions of these five topics were carefully reviewed by the author and they have been cited here for their reference value in providing the background which has contributed toward progressive deafness and otosclerosis.

In "Senile Deafness," Neville[39] reviewed the signs, symptoms and treatment of a group of 120 patients whose average age was 66.9 years.

The usual symptom was deafness alone; some complained of vertigo and tinnitus as well. In senile deafness, vertigo was considered to be usually due to a lesion in the central paths. In some it was due to changes in the semicircular canals.

Senile deafness started at the age of 50 years, with a decreased hearing at 2,048 cycles. The qualitative difference between the sounds of musical instruments was lost. These individuals recognized vowels as well as people with normal hearing but they did not recognize consonants so well. Consonants were complex and contained high frequency notes.

The difficulty in hearing when two or more people were speaking at once was a typical early symptom of senile deafness. According to Saxen, the pathologic changes in old age deafness were present in two forms: *a.* senile atrophy of the spiral ganglion; *b.* angiosclerotic degeneration of the internal ear. He related these pathologic findings to the clinical signs — thus, senile atrophy of the spiral ganglion produced the typical clinical symptoms of presbycusis. Angiosclerotic degeneration of the internal ear did not produce symptoms such as deafness until the sclerosis of the blood vessels had advanced so far as to produce degeneration of the organ of Corti and collapse of the cochlear duct.

Shortening of bone conduction, which is a prominent sign in senile deafness, was due chiefly to the disturbance in the production of endolymph and the collapse of the cochlear duct.

He felt that treatment was worth while and recommended, beside local treatment, the reduction of uric acid or cholesterin in the blood, the treatment of arteriosclerosis, certain drugs for vertigo and tinnitus, such as bulbocaprine, if the vertigo arose in the semicircular canals, and luminal if central in origin, and caffeine and luminal for tinnitus. He recommended vitamins, as suggested by Selfridge, and estrogenic hormones, as used by Koch.

Parker[40] reviewed the literature and presentations of cases of the syndrome of "Severe Uveitis with Associated Alopecia, Poliosis, Vitiligo and Deafness." Of 29 cases reported, temporary hearing impairment occurred in 69 per cent. In each case the loss of hearing came on at the same time as the ocular symptoms. The lesion was felt to be in the labyrinth or the central nervous system. The etiology was questionable. All the other lesions occurred in pigmented tissue and it was suggested that an accidental or congenital pigmentation of the basilar membrane may have occurred which underwent changes with resultant partial deafness. No specific treatment for this condition was available.

"Major Features in the Developmental History of the Human Stapes" were discussed by Anson.[41] He stated that the stapes differed from other bones in its development; it began as a ring and became stirrup-shaped, its full size being attained by very rapid growth in the midterm fetus. It had

attained adult dimensions while still a fetal bone. While long bones lacked osteogenic periosteum on their articular surfaces, the stapes lacked it on all surfaces. The stapes did not possess secondary centres of ossification at any stage of its development but developed from a single primary centre in its base. Only the tympanic extremity of the stapes was of true articular type — the vestibular extremity was articular marginally, the flattened surface closed a fenestral orifice in the otic capsule.

A masterful treatise on the morphology of the stapes, under the title, "Adult Form of the Human Stapes in the Light of Its Development," was presented by Beaton and Anson.[42] Their reasons for working on this problem were stated as follows: Without information upon the development and normal adult form of the stapes, pathologic alteration could not be distinguished from unimportant variations in size and shape, simple fragility from osteoporotic destruction, or normal histologic replacement of cartilage from the abnormal ossification of stapedial ankylosis. They stated that no single element in human anatomy had suffered more chronic neglect, and felt that textbook descriptions had become stylized and debased and were inaccurate and fragmentary. The article contained illustrations and accurate measurements with normal variations found.

In "The Endocrine Factor in Dysfunction of the Organs of Special Sense," Warwick[43] discussed principally the female sex hormone in relation to calcium and phosphorus metabolism and visualized the future treatment of squint, otosclerosis, muscular imbalance and cataract, as a routine of diet, vitamins and endocrine therapy.

(To be continued in June issue.)

LOCALIZED NONSUPPURATIVE ENCEPHALITIS OF OTORHINOLOGIC ORIGIN.*

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Localized nonsuppurative encephalitis is the rather high-sounding name that is given to a straightforward though not very common condition. It is part of the inflammatory process which occurs wherever infection enters the brain from an adjacent focus in the skull — whether nasal sinus, ear or open fracture.

The broad pathological basis of inflammation is the same no matter whereabouts in the body it occurs. Individual differences there are, differences of detail but not of principle, depending on the tissue invaded — the reaction of the glial elements in the brain, for instance. With these histological details, this paper is not concerned. The concern here is with the broad principle and its clinical recognition.

When infection invades the tissues — as a suitable example, the peritonsillar tissue — there is a mobilization of the body's defenses and an outpouring of leukocytes and serum which causes local swelling. According to the virulence of the infection and the resistance of the patient, either the inflammation subsides, or suppuration occurs in the centre, or in extreme cases no localization takes place and the infection spreads widely throughout the adjacent tissues, often causing necrosis, sometimes with a fatal result.

The process in the brain is no different. When infection from a nearby focus achieves the brain, an area of inflammation is produced, which in the majority of cases breaks down in the centre and forms the familiar brain abscess. But in a proportion of cases it does not break down. In these the swelling increases to a point, stops and subsides, in the meantime aping in its symptomatology the brain abscess of which it is so often the forerunner (see Fig. 1). This is localized nonsuppurative encephalitis as Oppenheim[1] described it in 1899, or pseudobrain abscess as Adson[2] dubbed it later, more

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graphically if less scientifically. There are a number of scattered case reports in the literature, but not until the last 20 years, with the work of Borries[3] and Symonds[4] has much interest been taken in this condition. Strangely enough, all the reported cases in the past except one (Behlau's[5]) have been in the temporal lobe. The temporal lobe is certainly the usual site for this condition, as it is for abscess, but in my 17 cases it is found also in every position in the brain and under all circumstances in which brain abscess arises, which, of course, is as it should be. A fuller report than I can give here will be found in my original paper.[6]

But this is only one of two varieties, the second of which has not been generally appreciated for what it is. The first

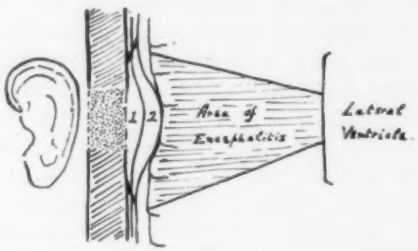

Fig 1. Diagram to show the spread of infection from ear to brain.

is the low virulence, subacute variety, the aborted abscess. The second is the high virulence, subacute variety, localized in that it is not diffuse throughout the brain but, all the same, spreading and not infrequently fatal, especially if necrosis of tissue takes place. These are the cases which in the past have been labeled acute abscess.

The clinical problem is to distinguish these two varieties of localized encephalitis from brain abscess, for on the correct evaluation of the state of affairs depends not only treatment but prognosis. In other words, when is a brain abscess not a brain abscess?

The difficulty of deciding when suppuration has taken place in an area of infection, when that area should be incised, is one which every surgeon has faced. To wait too long is to allow unnecessary destruction of tissue to occur and to sub-

ject the patient to equally unnecessary distress. On the other hand, to operate too soon is to interfere with the defense mechanisms already under way and perhaps to produce suppuration or extension of infection in an area of inflammation which might otherwise subside. Even in such an accessible position as has been taken as an example, the peritonsillar tissues, a position accessible both to observation and palpation, mistakes are made. There is probably no throat surgeon who has not incised a peritonsillar swelling in the expectation of finding pus, and been disappointed. The difficulty of decision is enormously greater in such an inaccessible position as the brain. Nevertheless, there are, even there, signs which, if carefully observed and correctly evaluated, will point the way to a correct decision.

1. Subacute Localized Encephalitis: In this group on the clinical side are the focal signs, often rather indefinite, of a lesion in an area of the brain adjacent to the infection, frontal, temporal or cerebellar, as the case may be. These signs moreover, are very changeable. A particular sign, as for instance an increase of reflexes on one side, which is present today may be absent tomorrow and present again the day after, a fact which makes it important not only to observe such cases day by day but to put down the observations made on paper so that memory is not assumed later to be at fault. This changeability of signs is very characteristic of a focus of infection in the brain and is caused by the varying amount of edema surrounding the focus. Alone, then, this come-and-go of signs is of no help in determining whether or not suppuration has occurred. The deciding factor lies in the findings in the cerebrospinal fluid.

The Cerebrospinal Fluid: The point of importance in the spinal fluid, the only point of importance in this connection, is the number of cells in the fluid. Just as in acute infections in other situations there is a massive early leukocytosis in the blood stream which diminishes as the infection becomes localized, so in the brain there is a preliminary local outpouring of cells to combat the infection, and this is reflected in a cell count in the cerebrospinal fluid in proportion to the virulence of the infection. If the condition is one of encephalitis without suppuration, the cell count is high, the actual number varying according to the virulence of the infection, but to

be counted in multiples of 10 — 50, 60 or even considerably higher. So long as suppuration does not occur, the count remains at about the same level or even rises, to fall as resolution or suppuration takes place. In the full-fledged localized abscess the count is low and to be counted in multiples of five — 20, 25 or so. The cells in the pure encephalitic case are mainly polymorphonuclears; in abscess cases, particularly if of long standing, mononuclears.

But the number of cells alone is inconclusive — suggestive rather than diagnostic. It is not until cell count is related to physical signs that the picture becomes clear. Borries[3] pointed out several years ago that an increase of localizing physical signs with a decrease of cells in the cerebrospinal fluid was characteristic of an abscess — what he called the "dissociation syndrome." This is in line with the process of suppuration elsewhere. The local manifestations of an abscess increase while the general signs decrease. On the other hand, if the infected area does not break down, the picture is that of an *association* syndrome — the cell count in the fluid increases up to a peak pari passu with the local signs, and the two subside together. And because the infection is not virulent enough to produce suppuration, it is not uncommon for the increase in cells to be slight — but at the same time so are the localizing signs.

Histology: The histological picture cannot be demonstrated, for a condition of making the diagnosis is that the patient shall recover. I have, therefore, no material. Presumably it must be the same as the area of encephalitis which is always present outside the capsule of an abscess and which is, so to speak, the remains of that in which the abscess is formed (see Fig. 2).

Summary: In sum. then, it appears that to distinguish between an area of infection in the brain which has broken down — an abscess — and one which has not, it is essential to make a daily observation of the patient, both as to focal signs and as to cell count in the cerebrospinal fluid, to set the two findings alongside each other and to note their relative progress. So long as the two keep pace with each other, increasing to a maximum and then subsiding, suppuration has not occurred.

Objection may be made that to do a daily lumbar puncture, or even a lumbar puncture at all if the lesion is in the pos-

terior fossa, is not unattended with risk. I do not believe this objection is valid. I am fully conscious of the dangers of lumbar puncture with posterior fossa lesions, but the amount required for a cell count is so small — and a cell count is all that is required — that the slight element of danger in the procedure is more than offset by the danger, by omitting it, of not arriving at a correct diagnosis. Of course, a large quantity of fluid must not be withdrawn, but 2 cc. is more than enough for the examination required.

Case 1. Localized Encephalitis, Right Temporal Lobe: A boy, age 13 years, was admitted to hospital with an acute

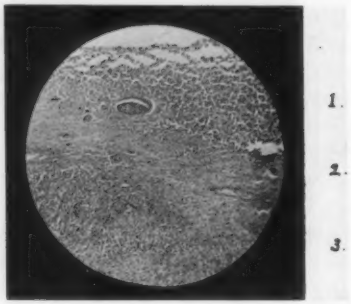

Fig. 2. The three layers of an abscess: (1) the inner granulation tissue; (2) the middle capsule; (3) the outer encephalitis. The demarcation by a capsular layer is apparent macroscopically as well as microscopically.

right mastoiditis and severe generalized headache. Pain in the ear had started five days before, headache on the morning of admission. No neurological signs were found.

Mastoid operation found widespread disease and a perisinus abscess. There was no exposure of middle fossa dura, either pathological or operative. The wound was left open.

Subsequent History: Six days later, he again complained of headache; temperature, 97°; pulse, 62. There were no neurological signs other than in the spinal fluid, which was under slight increase of pressure and contained six cells, all polymorphonuclears.

During the course of the next week his headache increased, he developed a mild left hemiparesis, he became drowsy, his

pulse fell to 54 on one occasion, his spinal fluid remained under increased pressure and showed a rising cell count, mainly polymorphonuclear — six, 14, 22, 182. 224.

Exploration of the right temporal lobe was undertaken on two occasions without result. After a week he began to improve, headache became less, hemiparesis improved, cell count in spinal fluid began to fall progressively — 170, 84, 22, five.

Result: The wound healed, all neurological signs disappeared, and he has had no recurrence of trouble.

Comment: This was 12 years ago. Now, with present knowledge, the typical picture of the association syndrome would not be overlooked and exploration would not have been undertaken.

2. Acute Hemorrhagic Encephalitis: It was said earlier that this variety of encephalitis is usually labeled acute abscess. When the physical signs of the two are compared it is difficult to understand how they could have been confused. The two have only one point of similarity — that there are signs of a focal lesion adjacent to the originating area of infection. But here all likeness ceases.

The outstanding characteristic of the patient with an abscess is his torpor. He lies quietly in bed in a state of semistupor from which he can be roused only with difficulty to answer questions, and into which he sinks again as soon as his tormenter ceases prodding him — or before. The onset of his malady is gradual and his focal signs vary in quality and quantity from day to day, sometimes disappearing for days at a time. His temperature and pulse tend to be subnormal.

The patient with acute localized encephalitis is in complete contrast. The onset is abrupt, frequently with a convulsion, he becomes restless, excited. even wildly delirious and may need to be restrained; his focal signs go on from strength to strength, always marked, never remitting until either he dies or starts to recover; his temperature and pulse are raised.

Cerebrospinal Fluid: The picture in the spinal fluid is also very different from that of an abscess. There is a great and

immediate increase in the number of cells, which may be counted in hundreds and, moreover, they are mainly lymphocytes. The association syndrome is evident also in these cases, cells and signs rising and falling together.

Histology: Since fatalities occur in this variety, the pathological picture is known. The brain shows an area of red softening, which, in the cerebrum, extends inwards to the lateral ventricle (see Fig. 3). In one case of mine there was

Fig. 3. Area of acute hemorrhagic encephalitis with some central necrosis. No limiting capsule.

found in this area patches of tissue necrosis without any evidence of localization or of true abscess formation. There was some perivascular hemorrhage and marked lymphocytic perivasculitis (see Fig. 4).

Summary: This group is easier of assessment than the previous one — rapid onset, great excitement, marked focal signs, a very high lymphocyte count in the fluid — these paint a picture which should be unmistakable. Such cases rarely form a localized abscess — either they resolve or they die, and the mortality rate is high. Half of my series of eight cases died.

Case 2. Acute Hemorrhagic Encephalitis, Left Temporal Lobe: A woman, age 36 years, was admitted to hospital on account of discharge from the left ear, severe frontal headache, dizziness and vomiting.

The history was that two weeks previously a myringotomy had been performed for an abscess in the left ear. She had had a varying amount of ear pain, headache and dizziness after this for four days, but all signs cleared and the ear dried after a week in hospital. Four days later the ear again discharged and all symptoms returned with increased vigor.

A mastoid operation was performed, and widespread disease found, with a pathological exposure of the dura, 0.5 x 2.5 cm., covered with granulations.

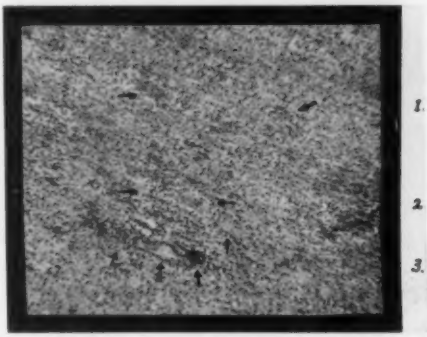

Fig. 4. Acute hemorrhagic encephalitis. Arrows point to: (1) thrombosed vessel with area of necrosis; (2) perivascular hemorrhage; (3) perivasculitis.

Subsequent Course: After the operation she became irrational at times, but there were no other neurological signs at first, and temperature and pulse were unremarkable.

Gradually she became more irrational and disoriented, at times violently delirious and had to be restrained; she developed a right hemiparesis, with aphasia when it could be tested; temperature varied around 102°, pulse around 110; while the cell count in the spinal fluid, which was consistently under pressure, rose from 234 to 614 to 4,000 eventually, with 50 per cent lymphocytes.

After the cells hit the 4,000 mark there was steady improvement, signs and cells receding together, and she was discharged a month later with all signs cleared except a slight residual aphasia. This was shortly before Christmas.

She was readmitted after Christmas with a recurrence of headache, nausea and dizziness. Aphasia was still present in slight degree, no worse, and no other neurological signs. Three days in bed put her right again and it was generally agreed that the diagnosis was Christmas festivities.

Encephalogram a month later showed slight ventricular pull to the left, indicating a scar on the left side. No sign of a space-occupying lesion.

Comment: Some aphasia persists, but all else is well. This was a very severe case, and an aphasia so deep as this patient had is very liable not to clear completely.

Treatment: Once the pathological process is appreciated and the diagnosis correctly made, the treatment, as usual, more or less settles itself. Above all things, these patients must not be explored. In the subacute variety, exploration may easily provoke the very thing it is wished to avoid — suppuration — but it has to be admitted that there are difficult cases in which the diagnosis can be settled in no other way. In the acute variety it may do worse — by encouraging spread, it may cause perforation into the ventricle. Three of my four fatal cases had been explored.

The first thing is to insure adequate drainage of the original focus, then to relieve pressure as necessary by internal and even perhaps by external decompression. In most cases repeated lumbar puncture when the lesion is above the tentorium, ventricular tap perhaps if below, combined with hypertonic saline or glucose is what is required. I have no experience of chemotherapy in this condition, but certainly in the acute cases it should be worth a trial.

3. Serous Encephalitis: There is a third variety of localized encephalitis which also leads to difficulties in diagnosis, the serous. No doubt the localized headache which is often complained of with a chronicly infected ear is due to such a serous encephalitis, but there is a more dramatic and more urgent form. Suddenly and without warning, a patient with a focus of infection in the skull is seized with a convulsion of Jacksonian type, which may be repeated and which usually leaves a slight paresis, which increases after each seizure. These cases of convulsions are due, many if not all of them, to an extradural focus which is often, but not always, an

abscess and which has produced a serous encephalitis beneath — an adjacent edema. The trouble clears rapidly if adequate drainage is secured promptly. I am firmly convinced that every patient of this type — with an infective focus in the skull, who is seized with a convulsion — demands immediate exploration of the focus as far as the dura, but in the first instance no further. Many of the cases reported of the onset of a brain abscess with a convulsion, I believe, follow on this condition, and that if promptly treated they would avoid the abscess. They are often "observed" until serous encephalitis becomes infective encephalitis and an abscess forms. Particularly does this happen with children. Children are supposed to be "given to" convulsions, and therefore the importance of the event is minimized and a waiting policy adopted. It is true that children are more liable to convulsions than adults, that where an adult would have a chill, a child will have a convulsion. Even so, there is a cause. Even the most provocative child will not throw a fit for no reason. It may be no more than a worm, but if there is a running ear it may also be a serous encephalitis.

Summary: The position may be summed up by saying that any patient who presents the association of a focus of infection in the skull with an otherwise unexplained convulsion of Jacksonian type deserves exploration of that focus as far as the dura.

Case 3. Serous Encephalitis, Right Frontal Lobe: A man, age 29 years, was admitted to hospital in a semiconscious condition after a fit.

The history was that 10 years previously he had been kicked on the forehead by a horse and had been unconscious for six hours. Three years later an exostosis was removed from his right frontal sinus. Eight years after that, and four days before admission, he had a severe headache over the right eye, followed the next day by a swelling. On the day of admission he had a fit, which started in the left hand and spread to arm, face and leg in that order, and became generalized. It was followed by five more with the same characteristics.

Examination showed a puffy swelling with indefinite fluctuation over the right frontal sinus, no pus in the nose. On

the neurological side there was left facial weakness of central type, all tendon reflexes were depressed. Left abdominals absent, both plantar responses were indefinitely extensor. There were no meningeal signs, the eye grounds were normal. Spinal fluid was under slightly increased pressure (220 mm.) and contained 86 cells, all polymorphonuclears.

Operation immediately found a fistula in the outer wall of the frontal sinus exuding pus, and another through the posterior wall, also exuding pus. A small extradural abscess was found, exposed to its limits and the wound left open. Recovery was uneventful, all neurological signs had disappeared in 48 hours, and there had been no recurrence when last seen 18 months later.

CONCLUSIONS.

1. Three varieties of this condition are described — two infective, subacute and acute — and one noninfective, serous.

2. Stress has been laid, as regards the first two, upon the importance of the cell count in the spinal fluid and its relation to the progression and regression of physical signs — the association syndrome.

3. While the mimicry of a brain abscess is very exact in the subacute variety, the symptomatology of the acute variety is entirely different, yet it is commonly diagnosed — and treated — as an acute abscess.

4. The importance of accurate diagnosis has been pointed out, in order that unnecessary and dangerous explorations be avoided.

5. In the serous variety, onset is frequently with a convulsion, and the importance of this event as an indication of extension of infection as far as the dura, and the danger of its extension *within* the dura, is discussed.

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123 East 61st Street.

SYMPOSIUM ON NASOPHARYNGEAL TUMORS.

(a) – THE PATHOLOGICAL DIAGNOSIS OF NASOPHARYNGEAL TUMORS.*

DR. ARTHUR P. STOUT, New York.

Tumors in the nasopharynx are not common. Portmann and Retrouvey report that they found only 32 nasopharyngeal tumors among 70,000 patients at the Bordeaux Otorhinolaryngologic Clinic. We may gain the same impression from the figures of Den Hoed, who found among 752 malignant tumors of the nose, throat and larynx only 47 in the nasopharynx. It is of interest to find that malignant growths are much more common than benign tumors. At the Presbyterian Hospital we have records of 82 cases of nasopharyngeal tumors and cysts; 23 of these were benign and 59 (72 per cent) were malignant. Dunlap called attention to the relative frequency of malignant tumors among the Chinese. He had seen 16 cases in private practice in Shanghai; all of them in Chinese, although only about 40 per cent of his patients were Chinese. Fourteen per cent of our malignant cases were in Chinese patients, although the hospital population averages only 0.3 per cent. All of their tumors were either epitheliomas or lymphoepitheliomas.

An analysis (lantern slide — table) of the types of growth found in our own group and in the reported cases shows that there are a few which are relatively common and a very considerable number which are so rare that they exist only in isolated case reports. Thus, after careful search, 34 different tumor types were found, but in our own collection only 12 of these are represented, and of these, only eight are represented by more than one example; therefore, only a few moments will be devoted to a discussion of the rarer lesions.

The neurilemoma is a solitary nerve sheath tumor which may arise in any place where nerves exist. It is always encapsulated. Hemangiomas usually occur in individuals with vascular tumors elsewhere. Lipomas are either polypoid or

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retropharyngeal. Xanthomas and amyloid tumors may both occur in the nasopharynx in individuals who do not suffer from generalized amyloidosis and xanthomatosis. The dermoid cyst, teratoma and epignathus are all due to congenital malpositions of tissue and usually appear in infants at birth or shortly thereafter. Epignathi are often very large and may project out of the mouth. They are composed of a complex conglomerate of differentiated tissues of various sorts and spring by a pedicle from the nasopharynx or lateral pharyngeal wall. The mixed tumor is entirely comparable to the mixed tumors found in other parts of the oral cavity, pharynx and salivary glands. Of the uncommon malignant tumors, it is of interest to note that leukemia, leukosarcoma, Hodgkin's disease, Kaposi's disease and sarcoid are simply local manifestations of generalized diseases. Chordoma and craniopharyngeal and hypophyseal rest tumors come from embryonal rests in the tissues above the vault of the nasopharynx. Hypophyseal adenoma in its anatomical position may grow downward by infiltration and manifest itself in the nasopharynx.

Turning now to the more common lesions, we find in the benign group, polyp, fibroma, cyst and chondroma. The polyp is just like the nasal polyp (Lantern slide 52687), a pedunculated, mucosal-covered, very edematous mass of fibroblastic tissue with relatively few blood vessels. The fibroma (Lantern slide 64873) has occasioned more interest and speculation because of its tendency to recur after removal and its usual occurrence in young males at puberty. It is composed of fibroblasts, reticulin fibres, which give it a firmer consistency than the polyp, and a great many capillary blood vessels. Unfortunately, this tumor has been reported under a variety of different pathological terms, such as angiofibroma, bleeding fibroma of masculine puberty, fibromyxoma and even myxosarcoma. It is not a malignant tumor, in spite of its tendency to recur and the fact that some patients have died of fatal hemorrhage after attempted removal. The cysts are simply retention cysts of the mucosal glands and seldom reach more than a centimetre in diameter. The chondromas (Lantern slide 64899) are benign cartilaginous tumors due, it is supposed, to proliferation of congenitally misplaced anlagen of Eustachian cartilage. They may form parts of polyps, as in this illustrated case, or the cartilage may grow within the

wall of the nasopharynx. They do not seem to reach a large size.

In order to understand the histology of the epithelial malignant tumors of the nasopharynx, it is necessary to recall that this cavity is lined in part by stratified squamous epithelium and in part by stratified cylindrical ciliated epithelium. It is provided with mucous and serous glands and, in places, with a great deal of lymphoid tissue. From the surface cells come epitheliomas of various degrees of differentiation, with most of them showing exceedingly little. Only four of our 29 epitheliomas showed marked epidermoid differentiation (Lantern slide 71211). Fourteen others showed the occasional formation of spherical bodies (Lantern slide 69218) resembling pearls but without keratinization or intercellular bridges, although the majority of the other cells were somewhat flattened. These tumors are classified as epitheliomas showing a slight degree of differentiation. Then other tumors (Lantern slide 62114) showed no definite epidermoid characteristics at all. They tended to grow in clearly defined cords of either rounded or slightly flattened cells. The inflammatory cells seen between the tumor cell cords in the lantern slide are often not present in this group and they do not intermingle with the tumor cells. This group we consider undifferentiated epitheliomas. It is, however, not easy to distinguish these undifferentiated epitheliomas from the so-called lymphoepitheliomas of Regaud and Schmincke, of which we have 16 examples. Very probably the lymphoepithelioma is simply a histological variant of the undifferentiated epithelioma. But, whatever its origin, it has morphological features which are distinctive enough to enable one to segregate it from the undifferentiated epithelioma on the one hand, and from the reticulum cell lymphosarcoma on the other — to both of which it bears some resemblance (Lantern slide 65089). The lymphoepithelioma is composed of anastomosing cords of completely anaplastic, irregularly rounded, large cells, which are clumped together without easily discernible cell membranes and no fibrous framework. The cords are embedded in lymphoid tissue and lymphocytes are often found scattered among the tumor cells. It requires a certain degree of experience before this tumor can be segregated with assurance but this is worth the effort because the results of treatment show that there is some biological difference between the lymphoepithe-

lioma and the other nasopharyngeal malignant tumors, as will be brought out by Dr. Kasabach.

The lymphosarcomas, of which there are 10 in our collection, may be roughly divided into the more common reticulum cell type, of which we have seven cases, and the lymphocytic cell type, of which we have three examples. The reticulum cell tumor (Lantern slide 61073) is a growth made up of cells considerably larger than the normal lymphocyte and more nearly approaching the size of the reticulum cell, or the germinal center cell of the lymph follicle. We do not know whether or not such tumors come from reticulum cells. The term is used because it has become current for tumors with this histological appearance. These cells are packed together in large masses diffusely and not separated into cords, as is the case of the epitheliomas. There is a delicate supportive framework but except for this there is no tendency toward pattern formation. The lymphocytic cell form of lymphosarcoma (Lantern slide 61966) has the same massive cellular infiltration or flooding of the tissues with cells, but in this instance the individual cells are smaller, not much larger than small lymphocytes.

Plasmocytomas are tumors of sometimes doubtful malignancy. Rarely the multiple myeloma of plasma cell type has a local manifestation in the nasopharynx. Usually, however, plasma cell tumors appear as solitary nodules or as multiple nodules in the upper air passages, with a tendency to reappear after removal. It is not possible to distinguish these two varieties by histological examination (Lantern slide 63460). The cells are crammed solidly together, as in the lymphosarcoma, with the characteristic eccentrically placed nucleus and homogeneous cytoplasm.

Finally, we must speak of the carcinomas. These are very rare. The only form with which I am acquainted is the glandular type which is usually called cylindroma (Lantern slide 44328). This is no doubt closely related to the so-called mixed tumor but differs from it in two important respects. In the first place, it is composed solely of cells which have a marked tendency to form acini instead of two or more different types of tissue, as in the case of mixed tumors. And, in the second place, it has a marked and definite tendency to infiltrate the tissues which surround it, instead of being confined within a capsule. The plugs of material which fill the tumor acini are

usually of a mucoid nature and are reddened by mucicarmine. This is a tumor which, in spite of its differentiated glandular appearance, is radiosensitive and it should always be treated by radiotherapy rather than by a futile attempt to excise it.

To recapitulate: In the nasopharynx, malignant tumors are thrice as frequent as benign tumors. Of the benign growths, polyps, fibromas and cysts are the most frequent, and of the malignant growths, epitheliomas, lymphoepitheliomas and cylindromatous carcinomas are most commonly encountered. There are 27 other tumor types recorded, but all of them are exceedingly rare.

NASOPHARYNGEAL TUMORS.

BENIGN	MALIGNANT PRIMARY	SECONDARY
Polyp 9	Epithelioma:	Leukosarcoma
Fibroma 4	well differentiated 4	Leukemia
Cyst (retention)..... 6	(epidermoid)	Hodgkin's disease
Neurilemoma..... 1	slightly differentiated 14	Kaposi's disease
Hemangioma	undifferentiated 10	Boeck's sarcoid
Lymphangioma	unclassified 1	Chordoma 1
Lipoma	— 29	Hypophyseal adenoma
Xanthoma	Lymphoepithelioma 16	Craniopharyngeal duct and hypophyseal rest tumors
Amyloid tumor	Regaud-Schminke)	
Chondroma 2	Carcinoma:	
Dermoid cyst	cylindroma 2	
Teratoma 1	solid type	
Epignathus	Malignant melanoma	
Mixed tumor	Lymphosarcoma:	
	reticulum cell..... 7	
	lymphocytic cell 3	
	— 10	
	Plasmocytoma 1	
	Fibrosarcoma	
	Sarcoma: undifferentiated	
	Rhabdomyoma	
	Malignant teratoma	

The table lists the names of tumors reported in the literature. The figures show the distribution of the 82 cases recorded in the Surgical Pathology Laboratory of Columbia University.

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630 West 168th Street.

SYMPOSIUM ON NASOPHARYNGEAL TUMORS.

(b) – OTOLARYNGOLOGICAL ASPECT.*

DR. JAMES W. BABCOCK, New York.

From a clinical viewpoint, the presenting symptoms of a patient with nonmalignant tumor of the nasopharynx differ greatly from those of one with a malignant growth in that region.

In nonmalignant tumors, the outstanding symptoms usually are bleeding and obstruction, with the secondary ear symptoms of deafness, tinnitus, otitis media or mastoiditis. Bleeding is particularly severe in fibromas and hemangiomas. These tumors were once a very serious problem as surgical removal was very difficult and attended by a great loss of blood; however, their ready response to radium, best applied by radon seeds, has lessened their terrors. Although nonmalignant, they may cause almost fatal loss of blood. I have even seen a young man with a fibroma whose hemoglobin was under 40 per cent, in spite of previous ligation of both external carotid arteries. In treatment of hemangiomas, injection of a sclerosing fluid in conjunction with radiotherapy is helpful. Pressure by any of these growths may cause destruction of surrounding parts but usually not to a serious extent, except to the ear. Here, as I have said, impaired hearing, tinnitus, acute otitis media and mastoiditis may result.

The malignant tumors arising in the nasopharynx are very insidious, frequently giving no subjective symptoms and reaching no great size at their site of origin. The patient's attention is usually called to them only when they have invaded the base of the skull or given rise to metastases in the cervical lymph nodes, so the length of time of their existence before such invasion of other structures occurs is problematical. In only one of the group of 42 from which most of these observations were made was the tumor discovered in a routine examination and, in spite of early treatment, it — a cylindroma — eventually caused the patient's death.

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Locally, obstruction to nasal breathing is commonly noted, often accompanied by some bleeding, usually not severe. Ulceration may be present with some discharge. Obstruction of the Eustachian tube is usually present, causing impaired hearing and tinnitus, varying in degree, going on at times to mastoiditis, requiring operation. This occurred in 10 per cent of this series. In one case a diagnosis of mastoiditis was made by radiographs when all that was present was a congestion and thickening of the lining of all the cells and middle ear, which cleared up, without operation, after treatment of the growth.

Cervical lymph node metastases are a very frequent presenting symptom, at times rather large, usually situated in the posterior triangle or under the upper third of the sternomastoid muscle, painless, hard and not breaking down. These are usually unilateral but may be bilateral, and at times the nasopharynx is not suspected until after their surgical removal. Stiffness of the neck is sometimes present with the enlarged nodes.

Trismus is present in a large number of cases, apparently due to invasion of the pterygoid muscles.

The symptoms due to invasion of the base of the skull vary greatly, depending on the parts involved. Headache is common. Change of personality may be present. The pathways into the skull are usually through the middle fossa and may attack the IInd, IIIrd, IVth, Vth and VIth cranial nerves, more commonly only the Vth and VIth, causing facial pain and diplopia from external rectus paralysis. Blindness, ptosis or ophthalmoplegia may result. On invasion through the foramen lacerum, the IXth, Xth, XIth and XIIth cranial nerves may be involved, causing dysphagia, hoarseness and paralysis of the tongue. The retroparotid sympathetic nerve may suffer, causing myosis and enophthalmos. The occipital bone and posterior fossa may also be affected. Not infrequently one or more of the above serve as the presenting symptom and an ophthalmologist or neurologist is consulted by the patient. So often is this the case that the usual rôle of the otolaryngologist in examining or treating a patient with a malignant nasopharyngeal tumor is searching for the already suspected tumor and removing a specimen for biopsy.

This is especially true since Schmincke's able description of the above symptoms in 1921.

Examination of the nasopharynx in these cases discloses variable findings. The tumor may greatly resemble adenoids with irregular surface, particularly in lymphoepithelioma, but is usually asymmetrical. In other cases the growth is smooth and occasionally ulcerated. At times nothing is seen but a small ulcerated area. A minority of cases have a large mass filling a large part of the nasopharynx and pushing the soft palate forward. Extension under the mucosa of the posterior pharyngeal wall down to the level of the epiglottis, with invasion of the vertebra, occurs but is usually not noticeable on inspection. Radiographs may show a shadow in lateral view, but are chiefly useful in disclosing destruction of the base of the skull when present.

The etiology seems to remain a mystery, but an unusually large proportion of the patients are Chinese, 20 per cent of this group. Three times as many males are affected as females. The duration of subjective symptoms, before patient's first visit, varied between two weeks and two years, and averaged six months.

Treatment is entirely in the hands of the radiotherapists, who offer the only hope for retardation or cure. Retardation is accomplished in a large percentage of cases, but cure, unfortunately, in very few. In the group studied, only 14.5 per cent could be considered cured for from one to five and one-half years. Of these, four were lymphoepithelioma, and two undifferentiated squamous-cell epithelioma; no sarcoma cures were obtained. The duration of life of patients dying is from one month to nine and one-half years, averaging 19 months. This does not include two cases who were admitted in extremis and not treated, nor one man who committed suicide shortly after treatment was instituted. The case dying nine and one-half years after first being treated had two recurrences after apparent cure in that period, the second one also temporarily yielding to radiotherapy. Dr. Kasabach has a table showing results obtained in the different types of malignancy of the nasopharynx, and, since many of the cases referred to by me are also in his list, I shall not duplicate his work. The behavior of all the types is uniformly bad, except

that better results have been obtained in lymphoepithelioma and undifferentiated squamous-cell epithelioma. The terminal cause of death may be pneumonia, meningitis, osteitis, which is apt to occur if subsequent trauma is inflicted on irradiated bone, or other possible complications, but no doubt is left that the neoplasm is the real culprit.

In conclusion, we must confess a disappointingly small amount of success with the early diagnosis and treatment of this very insidious and most malignant disease, even with the best of co-operation of the different branches of medicine into whose fields this neoplasm invades. We can only be thankful that its incidence is no greater and hope that the ever growing improvement in the technique of radiotherapy may solve this distressing problem.

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20 East 53rd Street.

SYMPOSIUM ON NASOPHARYNGEAL TUMORS.

(c) – FROM THE NEUROLOGICAL VIEWPOINT.*

DR. SAMUEL BROCK, New York.

In the last two decades considerable advance has been made in the differentiation of the finer histological characteristics of malignant growths in the nasopharynx. Moreover, the remarkable radiosensitiveness of the so-called lymphoepitheliomas and the squamous epitheliomas makes prompt diagnosis very important, the more so as these growths metastasize early and valuable time may be lost if one is not on the alert. All of this imposes a serious responsibility on the rhinologist, otologist, ophthalmologist and neurologist.

The benign tumors of the nasopharynx will not be touched upon here since they do not produce signs of neural involvement often, and because nasopharyngeal signs point the way to their presence.

Since 1922, the neural signs brought about by malignant tumors of the nasopharynx have been stressed by a number of investigators (New, 1922; Woltman, 1922; Worms and Carillon, 1930; Schlivek, 1932; Roger and Paillas, 1935; Wortis and Brock, 1936; and Needles, 1937).

Malignant tumors of the nasopharynx and, more especially, lymphoepitheliomas, may be divided into three groups from the viewpoint of *early* presenting symptoms and signs: *a*. Those in which cranial nerve signs are outstanding — about 50 per cent. *b*. Those presenting signs and symptoms referable to the naso- and oropharynx (as nasal obstruction, mucosanguineous discharge from the nose, middle ear deafness, stuffiness, and tinnitus due to Eustachian tube obstruction, dysphagia and recurrent aphonia) — about 30 per cent. *c*. Those first showing hard, shotty enlargement of the retro- or submaxillary lymph glands, on one or both sides — about 20 per cent. In the later stages of the disease, there is a commingling of these effects and these groups are no longer clearly defined.

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My remarks will be limited to the first group; *i.e.*, the cases with early neural signs. The latter are due to cranial nerve involvement brought about directly by the presence of neoplasm at the base of the skull, or less often by the pressure of enlarged lymph glands which are the site of metastatic invasion.

The nerves most commonly involved are the VIth and Vth nerves, which are implicated by the growth as it erodes the bones of the middle fossa of the skull. The involvement of the VIth nerve produces a partial external rectus weakness with diplopia on homonymous lateral gaze, which is in no way characteristic. Of far greater diagnostic significance is the trigeminal nerve involvement. Continuous or recurrent localized pain on one side of the face, or in or about the eye, or even in the mastoid region or external ear in a patient of *any age* should put one on guard — especially if there is an associated skin paresthesia, hypesthesia or anesthesia. in part or all of the domain of the first or second or third divisions of the Vth nerve with corresponding mucous membrane involvement. If there are some signs of motor trigeminal involvement, *i.e.*, ipsilateral wasting of the masseter and temporal muscles, and ipsilateral weakness of the pterygoid muscles, the opened jaw deviates to the affected side and one's suspicions should be increased. About 80 per cent of the cases belonging to this "neurological group" show involvement of the VIth and/or Vth nerves. This clinical picture has been called by the French "the syndrome of the point of petrous bone." The clinical picture should not be confused with paroxysmal trigeminal neuralgia (tic douloureux). In this latter affection, there are no objective sensory changes unless the branches of the trigeminal nerve have been injected with alcohol. Moreover, in tic douloureux, the pain is sharper, much more transient, and associated with trigger zones, stimulation of which induces the pain. In tic douloureux there is never any sign of involvement of the trigeminal motor root or of any other cranial nerve.

If the malignant process involves the posterior lacerated foramen, the IXth, Xth and XIth nerves suffer, and some of the following ipsilateral changes are found: loss of taste on the posterior third of the tongue, hemisensory defects in the soft palate, pharynx and larynx, one-sided paralysis of the soft palate, larynx, and sternomastoid and trapezius muscles.

Dysphonia and dysphagia may occur in these cases. Now and again, hemiatrophy and weakness of the tongue are added to the above signs, due to involvement of the skull farther posteriorly at the emergence of the XIIth nerve. In four of 35 cases, Needles noted the Horner syndrome (ipsilateral miosis and ptosis with enophthalmos) due to implication of the cervical sympathetic as it enters the skull with the internal carotid artery, through the medial lacerated foramen. In his series, Schlivek found signs of the Horner syndrome in 10 of 38 cases. Less commonly, the invasive process strikes the petrous-sphenoid region, to involve the optic and oculomotor (IIIrd, IVth and VIth) cranial nerves as well as the Vth, then some or all of the following changes are found: total or partial ophthalmoplegia, primary optic nerve atrophy and amblyopia or complete blindness, facial pain and motor and sensory trigeminal signs — all being on the same side as the lesion. Proptosis may also be present. The facial nerve is involved less often and the auditory nerve rarely, although ipsilateral middle ear deafness and tinnitus are commonly encountered, due to obstruction of the Eustachian tube.

Late in the disease, bilateral signs are not unusual. I will not refer to deformities of the orbit and bones of the face and head which may appear late. Evidences of increased intracranial pressure and papilloedema are not frequent until the very last stages of the disease have been reached.

Various authors have described signs referable to the spinal cord, due to late metastic involvement of this part of the central nervous system.

Finally, I would say that from the neurological viewpoint, a clinical picture referable to unilateral implication of the cranial nerves issuing from the middle or posterior fossa should make one search carefully for enlarged retro- or submaxillary cervical lymph glands. We must immediately enlist the aid of the rhinoscopist and insist on *repeated examinations of the nasopharynx.* Most of the time he can visualize the mass and obtain a piece by biopsy for tissue study. This will clinch the diagnosis and give valuable prognostic information. Study of the X-rays of the base of the skull for evidences of bony erosion (less often osteosclerosis) and of soft tissue masses encroaching on naso- or oropharynx is also of great value, as will be noted in Dr. Cornelius Dyke's paper.

1192 Park Avenue.

SYMPOSIUM ON NASOPHARYNGEAL TUMORS.

(d) – ROENTGENOLOGICAL ASPECTS.*

DR. CORNELIUS G. DYKE, New York.

This presentation consisted of lantern slides and their interpretation.

SYMPOSIUM ON NASOPHARYNGEAL TUMORS.

(e)—ROENTGENOGRAPHY OF THE MALIGNANT NASOPHARYNGEAL TUMORS. A PRELIMINARY REPORT.*†

DR. HAIG H. KASABACH, New York.

Between 1929 and 1939, 57 patients with malignant tumors of the nasopharynx were referred for treatment to the Radiotherapy Department of the Presbyterian Hospital. Of these, 42 were treated between 1929 and 1937.

For the purpose of this study, the 57 cases were divided into various histologic groups. The largest group consists of 29 squamous-cell epitheliomas, subdivided into eight undifferentiated, 17 slightly differentiated and four well differentiated types. Sixteen were classified as lymphoepitheliomas, representing the next largest group. The remaining 12 cases were divided into eight lymphosarcomas, three cylindromas and one plasmocytoma.

On admission, 44 of the 57 patients (77 per cent) showed clinical evidence of cervical lymph node metastases. In 23, the adenopathy was bilateral, and in 21 unilateral. In the unilateral lymphoadenopathy group, 14 were on the right, and seven on the left side.

Roentgenograms of the skull were taken in 28 cases where the patient's symptoms or the clinical signs on admission suggested possible extension of the malignant disease into the base of the cranium. Of these, 17 (30 per cent) revealed Roentgenographic evidence of invasion of the basilar bones or adjacent sinuses. Distant bone metastases occurred in four cases in the late stage of the disease.

It is of unusual interest to note that nine of the 57 patients were Chinese. This observation of high incidence of malignant tumors of the nasopharynx among Chinese is in accordance with that reported by A. M. Dunlap, of Shanghai.

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The result in 42 cases of various types of malignant nasopharyngeal tumors treated between 1929 and 1937 are shown in Table I. The majority of these patients received long series of fractionated doses of Roentgentherapy to the nasopharynx through several portals. In 14 of the 16 squamous-cell epitheliomas there was noted an immediate response to radiation and primary disappearance of the disease soon after the termination of the treatment. The results, as shown in Table I, were more striking in the lymphoepithelioma and lymphosarcoma group. Of the 42 patients, 27 are dead. Twenty-two died in less than three years; two died after three years, and the remaining three died after surviving five years or longer from the date of admission. Further analysis of the cases who died after surviving more than three years reveals that the cause of death in one patient was ascribed to cardiovascular disease. Another one died of advanced pulmonary tuberculosis, and the remaining two died of reappearance of the disease with metastases. There are 14 cases who survived over three years with freedom of disease; seven are alive and well five years and over. In this survival group it is of interest to note that the majority of them are lymphoepitheliomas.

Because of the rapidly metastasizing nature of the majority of the nasopharyngeal neoplasms and their inaccessibility, we have learned the futility of local measures, such as surgery, cautery, topical or interstitial application of radium or radon. The type and technique of the treatment we employ are summarized in Nelson's Loose Leaf Surgery by Lenz and Haagensen in their chapter on "The Treatment of the Regional Forms of Cancer". We rely mainly on intensive, fractionated Roentgenography to the side of the primary tumor and usual routes of extension. It goes without saying that prior to the treatment, a thorough physical examination is done and oral and dental hygiene is carried out.

The daily dosage of radiation is regulated by the patient's general condition, his reaction to the treatment and the extent of the disease. Thus, the treatment may be purely palliative or, where conditions warrant, radiation dosage may be extended to the limit of the patient's tolerance. The principal fields used are graphically demonstrated in Table II. Since the nasopharyngeal neoplasms frequently extend up-

ward, invading the base of the cranium, it is important to use lateral portals sufficiently large to include the base of the skull. More dosage into the depth of the tumor can be added by irradiating the tumor through various directions. Lymphosarcomas as a rule require relatively less irradiation and may be eradicated by a smaller total dose. It should be emphasized that, in spite of primary, rapid regression or disappearance of the tumor during the course of irradiation, treatment should not be suspended before the entire dose has been given, otherwise the disease may reappear soon. Furthermore, rapid necrosis of the tumor should be avoided because of the possibility of consequent absorption of toxic products and injury to the patient's general well-being. This is particularly true in lymphosarcoma. In the beginning of the course it is desirable to administer daily small "test" doses and continue the subsequent course according to the response of the patient.

From this brief presentation it is shown that the majority of the nasopharyngeal tumors are highly malignant, and the results are far from being satisfactory. It would be of great practical value if one could enumerate the causes of failures in the unsuccessful cases. The inadequacy of radiation, the anatomical location and microscopic character of the growth give us some clue. It has been pointed out that the majority of the neoplasms are of low degree of differentiation. On the basis of our experience, the lymphoepithelioma group has shown a far more favorable prognosis for longer survival than any other group. One of the most important factors in all unsuccessful cases was the presence of bilateral cervical metastases. The majority of these patients died within a period of one year; whereas the cases who are still alive and well over three years had a few clinically palpable nodes or none at all. It may be added that, with a few exceptions, the presence of bone destruction has proved to be a grave prognostic sign.

CONCLUSIONS.

1. The malignant tumors of the nasopharynx are as a rule highly radiosensitive.

2. Because of the rapidly metastasizing nature of these lesions and their inaccessibility, adequate Roentgenography

TABLE I. MALIGNANT TUMORS OF THE NASOPHARYNX

(ROENTGENOGRAPHY) — 1929-1937.

3 and more yrs. Follow-up.

Histological Classification	No. of Cases	Primary Healing	Died				Living with Disease		Living — Well		Total		Remarks
			Less than 1 yr.	2 yr.	3 yr.+	5 yr.+	3 yr.	5 yr.+	3 yr.+	5 yr.+	Died	Living and Well	
Squamous-cell epithelioma:	16	14	5	5	1	1	....	....	3	1	12	4	*Cause of death—Pulm. T. B.
(1) Undifferentiated	4	4	2	....	....	....	....	....	2	....	....	....	
(2) Slightly differentiated	9	8	1	5	1*	1	....	....	....	1	....	....	
(3) Well differentiated	3	2	2	....	....	....	....	....	1				
Lymphoepithelioma	14	14	2	3	....	1	....	....	4	4	6	8 (57%)	
Lymphosarcoma	8	7	4	2	....	....	....	....	....	2	....	2	
(1) Reticulum cell type	6	5	4	1	....	....	....	....	....	1	....	....	
(2) Lymphocytic type	2	2	....	1	....	....	....	....	....	1	6	....	
Cylindroma	3	3	1	....	1*	1**	....	....	....	....	3	....	*Died of Ca. of U-bladder **Died of heart disease
Plasmocytoma	1	Slight	....	....	....	....	1	....	....	....	....	....	
Total	42	38	12	10	2	3	1	....	7	7 (16.3%)	27	14 (33%)	

is the only reliable procedure by which to attack the disease.

3. In the majority of instances a total disappearance of the neoplasm, including the regional lymph node metastases, can be achieved. This may be temporary or permanent, depending on the histogenesis of the tumor, the patient's general condition and the extent of metastases.

4. The results in 42 cases that we have treated between 1929 and 1937 and followed three or more years show that 14 (33.3 per cent) are alive and well. Of these, seven are clinically free from disease over five years (16.6 per cent).

5. Further progress in this field may be expected when more attention is devoted to early recognition of the primary lesion and adequate Roentgentherapy.

TABLE II.

These are the principal fields of external radiation used for the treatment of malignant nasopharyngeal tumors:

No. 1 and No. 2 — Lateral portals include the base of the skull and nasopharynx.

No. 3 and No. 4 — Antral fields used to add more dosage to the depth of the tumor. One large field may also be used. Both eyes should be shielded.

Nos. 5 and 6—These portals are used when the regional nodes are involved. Additional portals may be used, depending on the extent of the metastases.

622 West 168th Street.

ACUTE OSTEOMYELITIS OF THE SUPERIOR MAXILLA IN CHILDREN. CASE REPORT.

DR. H. D. HARLOWE, Virginia, Minn.

Necrosis of the upper jaw in its acute form differs in no way from osteomyelitis in general except that it is a disease peculiar to newborn and young infants. Only rarely does osteomyelitis of the superior maxilla occur in adults; then usually as a secondary complication. In a study made by Wilensky,[1] 82.5 per cent of the cases occurred within the first two months of life. Skillern[2] states that this form of osteomyelitis is seldom found after puberty. According to Hajek,[3] idiopathic osteomyelitis of the superior maxilla is the most uncommon type and occurs only in infancy and earliest childhood. Similar are the findings of Lacy and Engel,[4] which, in addition, reveal the rarity of the disease by approximating only 70 cases to have been reported since 1847. All statistics, however, present a clinical picture which is strikingly uniform in the course of the disease.

REPORT OF CASE.

History: J. B., a white female child, age 6 years, was first seen in February, 1939. Her mother states that three weeks ago the child had had an attack of influenza. Subsequently she noticed a swelling of the left cheek, a slight discharge from the left nostril and loosening of the upper teeth on the affected side. A tentative diagnosis of left acute maxillary sinusitis with probable acute osteomyelitis of the left maxilla was made. The patient was hospitalized for further study and treatment.

Past and Family History: Irrelevant.

Physical Examination: The general appearance of the child was pale and undernourished. The left side of her face was swollen, including the lower eyelid. The left nostril was filled with purulent discharge, which appeared to come from beneath the inferior turbinate. The right nostril was free of discharge. A small discharging fistula was present in the

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left superior alveolar process. The mucosa over the hard palate of the affected side was markedly swollen and cyanotic. All teeth in the corresponding region were loosened. The tonsils had been removed satisfactorily.

Laboratory Findings: Hemoglobin, 60 per cent; red blood cells, 3,800,000; white blood cells, 11,700. The differential blood count revealed 65 per cent polymorphonuclears, 31 per cent lymphocytes, 3.5 per cent monocytes, and 0.5 per cent

Fig. 1. Patient with acute osteomyelitis of the left superior maxilla.

eosinophils. Wassermann and Mantoux tests and the urinalysis were all negative. Bacteriological study of the discharge from the nose and discharging fistula revealed staphylococcus aureus. Biopsy report: Chronic inflammatory changes of the left maxillary sinus. Roentgenograms of the accessory sinuses taken on admission revealed a cloudiness of the left antrum. No evidence of osteomyelitis could be made out at this time. Subsequent Roentgenograms confirmed the diagnosis of osteomyelitis of the left superior maxilla with sequestra.

Treatment and Course: The patient's temperature on admission to the hospital was 39.5° C. (103° F.). An intranasal antrotomy and dilation of the alveolar fistula was performed under a general anesthesia. Purulent discharge and necrotic tissue were encountered in the left maxillary

sinus. Specimens were taken for biopsy and bacteriological study. The patient received sulfanilamide in the appropriate amounts. The maxillary sinus was irrigated frequently with warm physiological salt solution. A diet high in vitamins and calcium was recommended. The patient's hemoglobin was improved under an internist's care. Removal of four loose teeth and sequestra was carried out periodically. The amount of necrosis of the superior maxilla is shown in Fig. 1. Four months following the onset of the patient's illness a large sequestrum (see Fig. 2) fell out spontaneously. Her

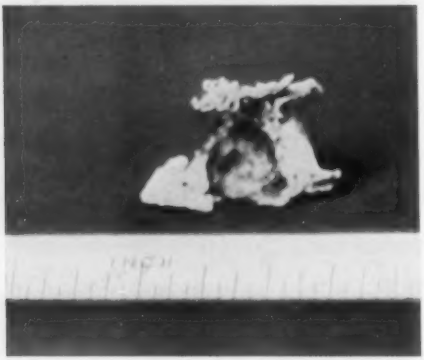

Fig. 2. Large sequestrum from the left superior maxilla.

condition improved readily, and she was referred to a dentist for future dental observation and treatment. A photograph taken of the patient one year following the illness is shown in Fig. 3.

COMMENT.

In dealing with acute osteomyelitis of the maxilla, most authors note a marked similarity in the early symptoms and physical findings. This fact is substantiated by the observations of Forbes,[5] Goldbloom and Bacal;[6] however, the etiology of the disease is as yet a matter of varying opinions. The investigations of these cases by Swoboda,[7] Zarfl,[8] Galli[9] and Remky[10] revealed an origin which was hematogenous or odentogenous in character. Somewhat opposed to this view

were the deductions of Posey,[11] Bronner,[12] Paunz,[13] Poncher and Blayney[14] and Lederer,[15] who believe that nasal infection and sinusitis may be the causative factor. Studies made by Fleming,[16] Van Gilse,[17] Mithoefer[18] and Jervey[19] conclude that the pathogenesis of osteomyelitis of the maxilla may be variable.

In treating this condition, the necessity for conservative measures whenever possible is strongly recommended. The establishment of adequate drainage through the endonasal or canine fossa approaches is desired. Lillie[20] is of the opin-

Fig. 3. Patient one year following illness.

ion that drainage in these cases is often undertaken too soon and that better results may be obtained by delay until the suppurative process is definitely localized. Schaeffer[21] has pointed out that the operator must always remember that the rudiments of both the deciduous and permanent teeth are present in the maxilla at the time of birth. Any injury to a "tooth germ" may lead to death of the part or the ultimate eruption of a deformed tooth. Not until after the fifteenth year does the maxillary sinus, in a sense, pass into the adult stage.

It has been suggested that avitaminosis may be a contributing factor in the pathogenesis of osteomyelitis of the maxilla. This entity impresses one with its relative infrequency and high mortality rate, which, according to Terracol,[22] may

vary from 30 to 50 per cent. The course of this disease is usually lengthy. This case extended over a period of eight months. A similar case reported by Iwamoto[23] lasted more than one year.

SUMMARY AND CONCLUSIONS.

1. Acute osteomyelitis of the superior maxilla confines itself primarily to nurslings and infants.

2. A case of this disease occurring in a child, age 6 years, with recovery following conservative treatment is reported.

3. Opinions are divided on the pathogenesis of this disease.

4. The possibility of injury to the deciduous or permanent teeth when operating on the maxilla in infants and children must always be kept in mind.

5. Avitaminosis as a possible contributing factor in the pathogenesis of osteomyelitis of the maxilla is mentioned.

6. The course of this disease is often lengthy.

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IN MEMORIAM

PROFESSOR MARCUS HAJEK

1861 - 1941.

We have recently received the sad news of the passing of Prof. Marcus Hajek, who died in exile in London on April 3, 1941. He left his home in Vienna, where he had achieved fame as a physician, teacher and scientist, two years before his death, and went to London as a guest of Sir St. Clair Thomson, who had invited him in the following words: "As long as God wills and as long as you are happy in my company, I should like to have you and your wife in my home."

A short review of his life and accomplishments should be of interest to his numerous pupils and colleagues in all parts of the world. His life was a difficult battle with many victories; it was the life of a self-made man of unusual capacity.

Marcus Hajek was born Nov. 25, 1861, in a small village in Hungary, which later was incorporated into Jugo-Slavia. His father was a poor itinerant peddler, and Marcus, carrying a heavy pack, accompanied his father in his wanderings from village to village. At the age of 13 years, supplied with a railroad ticket and a morsel of bread, the boy, Marcus, left his home and went to a neighboring town to enter high school. From this time forth he made his own way.

After graduating from high school, he began the study of medicine in Vienna without any knowledge of the German language. His only book was a German translation of Shakespeare. He acted as tutor for medical students. During this period of his life he was invited to the home of Prof. Schnitzler, the founder of the Vienna Nose and Throat Clinic. Subsequently he was married to Prof. Schnitzler's daughter, Gisela, much against the wishes of her father.

Later he became assistant in the Polyclinic and published a number of outstanding articles on the paranasal sinuses. He had great difficulty in obtaining the appointment as a docent and was rejected twice by Prof. Schroetter. Upon his

third appearance before the latter he presented himself with an additional number of publications, but Schroetter said, "As long as I live, I shall not appoint you," to which Hajek in characteristic fashion replied, "Then, Professor, it would be dishonest for me to wish you a very long life." He then turned on his heels and walked out, but was appointed in the same year.

In the subsequent decades he was exceedingly active as a scientist and teacher, and hundreds of physicians, among whom were many Americans, attended his courses in Vienna. Many of his students recall the remarkable collection of anatomic and pathologic specimens which he, himself, had prepared. In 1919 he became chief of the University Clinic, and he held this position until he had attained the age of retirement, 71 years. At one time he was given the title of Honorary Citizen of Vienna.

It is not difficult to evaluate Hajek's distinction as a teacher. His chief characteristic was in the personal example which he set for his pupils and colleagues, especially in the strict scientific standards which he set for himself. His dictum was, "Learn to find what you see and not to see what you wish to find." The period of years that he spent as chief of the clinic was among the most fruitful periods in the development of the field of rhinolaryngology. The number of his publications is enormous, and much of his work was fundamental. For example, in the Handbook of Denker-Kahler there are 316 references to his name. His well-known textbook on the pathology and therapy of the nasal sinuses is an internationally accepted standard book and has been translated into almost all languages. In 1932, his precise and remarkably lucid textbook on the diseases of the larynx, trachea and bronchi was published and received universal acclaim.

Hajek was a many-sided man. After his retirement as professor, he took up the study of piano and the study of physics. His keen humor, sometimes tempered with sarcasm, was well-known to his students. This is well illustrated by an anecdote

which occurred just before his departure from Vienna. He was asked by an official, "Why do you leave Vienna? Nothing harmful will happen to you." Hajek replied, "If you will keep my confidence, I will tell you the truth. I have committed many sins in my life, and when I die I undoubtedly will go to hell, but I do not wish to enter hell before my demise and, therefore, I am leaving Germany." However, those who know him well are convinced that he has received the beneficent eternal reward which he so richly deserved and that his fame will live after him. K. T.

DAYTON C. MILLER, M.D.,

1867-1941.

The Case School of Applied Science has suffered a great loss in the death of Dr. Dayton C. Miller, on Feb. 22, at the age of 74 years.

Dr. Miller had taught at Case for more than 50 years and had been head of the Department of Physics from 1893 until his retirement as Honorary Professor of Physics in 1936. In 1927 he had been made Ambrose Swasey Research Professor of Physics. Upon his retirement in 1936, the Case Trustees bestowed upon him the title, "Honorary Professor of Physics" for life, and at their request he continued as acting head of the department until the fall of 1939. He was an Honorary Member of the American Otological Society since 1925.

Internationally known for his work in quantitative measurements of light and sound and as the inventor of the Phonodeik, which turns sound waves into a moving beam of light upon a screen, he was at the same time a modest and lovable teacher, always searching for the truth and ever interested in the problems of his students.

Dr. Miller's work in the field of acoustics was widely recognized. He studied acoustics in relation to auditorium design and drew the specifications for some of the large auditoriums throughout the United States.

Another achievement was in his research on "ether drift," in which he sought proof of the earth's motion through the ether of space. This research followed the famous Michelson-Morley ether drift experiments of 1887, which originated on the Case campus. Dr. Miller's ether drift studies, beginning in 1901, were brought to conclusions on a positive drift, and his paper was presented before the National Academy of Sciences meeting at Princeton University on Nov. 18, 1929.

For three years Dr. Miller was Chairman of the Division of Physical Sciences of the National Research Council of Washington. Since 1914 he was a member of the Council of the American Physical Society, and was President in 1925-

1926. He served as Secretary of the Physics Section of the American Association for the Advancement of Science in 1902-1906, was Vice-President in 1907, was Secretary of the Council in 1908, and General Secretary in 1909. Besides these societies, Dr. Miller was a member of 33 other organizations whose interests lay in the fields of physics, astronomy, acoustics, optics, engineering education, mathematics, scholarship and music. His hobby was that of collecting rare flutes and old manuscripts of flute music. There are now 1,400 flutes in the collection, the largest in the world, and they are of silver, gold, glass and wood, many of them very old. The one he made himself is of 22-karat gold and has such a superior tone that it frequently is used in symphony concerts.

Dr. Miller graduated from Baldwin-Wallace College with the degrees of Bachelor of Arts in 1886 and Master of Arts in 1889. In 1890 the degree of Doctor of Science was conferred upon him by Princeton University. Since then he has received five honorary degrees: Doctor of Science from Miami University in 1924; from Dartmouth in 1927; Doctor of Law from Western Reserve University in 1927 and the same from Baldwin-Wallace in 1933. Case School of Applied Science conferred the Degree of Doctor of Engineering in 1936.

In 1917, and again in 1927, two medals were presented to him by the Franklin Institute. In 1925, he won the $1,000 prize of the American Association for the Advancement of Science, and the city of Cleveland gave him the medal for distinguished service in 1928.

Dr. Miller had the distinction of having made the first surgical X-ray photograph in America. This was for Dr. George Crile, now the head of the Cleveland Clinic, and one of the many who paid tribute to Dr. Miller's greatness at the time of the funeral on Feb. 24 at the Euclid Avenue Congregational Church, Cleveland, Ohio, of which Dr. Miller was a member.

"Dayton Miller has been distinguished by his high intelligence, his originality, his power of logical thinking and the

force of a unique personality. . . . Judged by every criterion, Dayton Miller has been one of the great men of his day," said Dr. Crile in his tribute.

Dr. Miller's wife, Mrs. Edith Easton Miller, to whom he had been married for nearly 48 years, survives him.

H. R. Y.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Regular Meeting, Nov. 20, 1940.

Surgical Aspects of Carcinoma of the Larynx. Dr. John D. Kernan.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Roentgenotherapy of Cancer of the Larynx and Pharynx. Dr. Maurice Lenz.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Lantern Slide Demonstration of Portmann Operation. Dr. George R. Brighton.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. RUDOLPH KRAMER: Up to 10 years ago, before the advent of radiotherapy — and by radiotherapy I refer entirely to the fractional dosage of Coutard, such as Dr. Lenz and others employ — I felt, as some men still do today, that laryngectomy must be done to cure carcinoma of the larynx. With the advent of radiotherapy and the establishment of laryngofissure, I changed my views. It is now simply a question of deciding how we are going to treat these cases. Certainly the results of laryngofissure in the strictly delimited cases are excellent, the five-year cures ranging all the way up to the figures quoted by Dr. Kernan of 85 per cent. But the limits are extremely sharply defined and even today, with the greater attention paid by the populace and the medical profession to the causes of hoarseness, we see comparatively few early cases.

It is true that the majority of cordal lesions start on the middle or anterior third of the vocal cords, but every once in a while, contrary to the statement of Dr. Hays Martin, the posterior portion of the cord is involved, even the inner surface of the vocal process of the arytenoid. Here, the possibility of doing a satisfactory laryngofissure is out of the question and, in contradistinction to Dr. Lenz' statement on this point, I know of several cases in which patients treated by radiotherapy for this lesion have been cured. There you see just one illustration of three neighboring institutions within five miles of one another having different experiences and results. As I said two years ago when I reported on this subject, we will have to get a great deal of experience and a large number of cases before coming to conclusions, but I still feel as I did then, that I have certain clues as to the indications for the method of treatment.

As I see it, the problem of the use of radiotherapy or surgery (laryngofissure) in cases with the indications for this surgical operation as laid down by Dr. Kernan is mainly an extramedical problem. This means that radiotherapy, properly applied, requires five to six weeks of treatment; the patient is uncomfortable; he must have a daily treatment, exclusive of Sundays and holidays, which implies constant attention by the radiotherapist; and the patient's discomfort, which is pretty marked, continues for four to six weeks in general. It is an expensive procedure. On the advantageous side, the patient generally comes out with either a perfectly normal or a fairly good voice. On the other hand, if you do a laryngofissure in these cases, the patient is generally through with the operation in a week and out of the hospital. It is much less expensive and the patient is much less uncomfortable. The voice,

in a majority of cases, is not nearly as good as that following radiotherapy, but that is a matter of minor importance, of course; however, I think the number of cures by surgery in these limited lesions is somewhat larger than by radiotherapy, perhaps 10 per cent. So, from these standpoints, which are mainly extramedical, I would say that limited vocal cord lesions are generally better treated by laryngofissure.

There are some patients in whom the indications for treatment were set by different considerations and it is from these patients that we got our experience and learned the results of radiotherapy. These patients had general medical conditions which made operation hazardous and for that reason were not operated upon. They were treated by radiotherapy, and did very well. So that I should say that the main advantages lie with laryngofissure, surgery, but that general and extramedical conditions must be kept in mind.

As far as the relationship between total laryngectomy and radiotherapy is concerned, I would not give a patient with a subglottic lesion radiotherapy but I would do a total laryngectomy. Outside of that, any laryngeal lesion for which total laryngectomy is indicated should be treated by radiotherapy first. I have had this experience: some cases treated by radiotherapy were not cured and then came to laryngectomy. There is a belief in some quarters that cases once radiated cannot be operated upon without great dangers of necrosis, infection and death. I have had almost as good postoperative healing following operation as if radiation had not been used. These results depend on the minutiae of the technique of the Roentgen ray application.

I have also had this experience: A patient had an extensive glandular involvement with a spread of the lesion from the larynx into the pyriform fossa and up along the side of the neck, an apparently hopeless case in which even radiation was not expected to help. But the patient was radiated and the glands and the pyriform fossa lesion disappeared, leaving what we interpreted as mother cells in the larynx. Then a total laryngectomy was performed and the patient is well and free of disease.

Another point as far as the treatment of laryngeal malignancy by radiotherapy is concerned: every once in a while you will find that the larynx clears up and then some years later a metastasis in a distant gland appears, perhaps supraclavicular or high up under the jaw. In some cases these have been treated by radiation and have disappeared. I have three such cases now in which the patients are well three years or more after the second course of radiation which was applied to metastatic glands.

I think that if we pool our experiences, try to employ a more or less uniform technique, use similar indications and describe our cases carefully, we will get somewhere after a period of years. Certainly fifty or a hundred isolated cases are of limited value. The techniques especially are not comparable. There are a number of experiences I have had that do not coincide with those of Dr. Lenz or Dr. Kernan — for example, the difficulties after doing a tracheotomy. We have a number of cases in which we have tried to estimate whether the lesion was so extensive that tracheotomy would be necessary before the end of radiotherapy, or whether the prospects were fair that the patient might escape tracheotomy. We make the tracheotomy very low, just over the sternum and out of the field of radiation. The outcome in these patients has not been altered materially by tracheotomy.

In some cases I have found combinations of surgery and radiotherapy extremely effective. Sometimes the one, other times the other is used first. In this way, cures have been obtained which were not possible when one or the other type of therapy was used alone.

Finally, we have not found that the histologic picture gives any indications as to the prognosis or the type of therapy that will give the best results. When I say that the results after radiotherapy have no connection with the histological picture, I am speaking only of the various types and grades of squamous cell carcinoma.

(To be continued in a succeeding issue.)

BOOK REVIEW.

Doctors in Shirt Sleeves (Musings on Hobbies, Meals, Patients — Sport and Philosophy.) Edited by Sir Henry Bashford. Veritas Press, New York.

To quote from the Editor's foreword, this volume was compiled from the weekly feature of the Lancet. Much of the subject matter has been obtained from the above columns, entitled, "Grains and Scruples." Sir Henry tritely states, if there should be any readers who believe that doctors as a race are myopic in their worship of science and quite unaware of its limitations, they should turn to the pages written in this volume by its numerous contributors, and readily learn that surgeons are not mere carpenters; and fishermen, gardeners and mountaineers could find much in common with the writers on sympathetic subjects."

In an interesting and intimate chapter on "Crabbed Age and Youth," by W. Langdon-Brown, the author humorously refers to a member of the "consulting staff" of a hospital as a position of great dignity but of no importance. He recalls his experiences on the active staff, and his efforts to be particularly polite to such consultants as he thought they were dead without their knowing it, until he reached a similar status, when, to his surprise, he felt that he was still very much alive; in fact, in some respects more so. He mentions numerous incidents in his 50 years' experience in a humorous manner, and depicts the many changes that have taken place. He ends his interesting chapter with the following thought, "Be not afraid of life. Believe that life is worth living and your belief will help create the fact."

L. N. Jackson's chapter is entitled, "Some West Country Patients," and gives his amusing experiences with types of individuals who prove so many things happen not according to the book, and that there is nothing like medicine for keeping a man humble.

Other chapters are: "From the Back Street," by F. G. Layton, J.P.; "A Little Learning," by A. A. Miles; "The Life of a Surgeon," by W. H. Ogilvie; "Pity the Poor Physician," by H. A. Dunlop; "On Hobbies and Old Age," by Louis Smith; "Too Much Prevention," by Lindsey Batten; "Some Thoughts on Meals and the Weather, etc.," by H. E. Boycott; "Some Public Memories," by T. B. Layton; "Toys and Some Other Things," by R. McNair Wilson; "The Biology of War and a Word on the Modern Woman," by Stephen Taylor; "Medicine and Philosophy," by Kenneth Walker; "Some Old Days in India," by Sir David Monroe; "Science and Spiritual Values," by R. S. Aiken; "Retirement and a Garden," by Philip Gosse.

In the last chapter, the author asks the question, "What are the rewards for gardening?" His answer is a thought to be borne in mind by other colleagues, especially by those who had lived their lives in large cities. "Health, a happy, contented mind, and a pleasant sensation of successful achievement, a lovely garden to surround his home, and, incidentally, an absorbing subject to discuss with his patients and so distract them from their minor symptoms."

The hobby of gardening is a particularly happy choice for a member of the profession, for in his retirement he will have a pleasant occupation to pass his time usefully and happily.

The volume is interesting, well written and splendidly typed — worthy of a place in any library and especially in the doctor's. M. D. L.